



SIR GORDON GORDON-TAYLOR, K.B.E., C.B., M.A., M.S., M.B., B.Sc., F.R.C.S.

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SIR GORDON GORDON-TAYLOR

GORDON GORDON-TAYLOR was a man whose life was completely dedicated to surgery. There have been many dedicated surgeons, some to the perfection of their own skills, some to the teaching of surgery and some to their patients. Gordon-Taylor was all of these but in addition he possessed a deep and enduring interest in the young surgeons in the decades behind him, particularly those in the formative stages of their careers. Wherever he travelled in the world it was these young men whom he would seek out and to them his help and advice was always freely given. After giving up active surgery himself, he devoted part of each week in his office at the Royal College of Surgeons, to be available to those who sought his help. His knowledge of the progress in life of the younger generation of surgeons was encyclopaedic and his continuing interest in their welfare never failed.

Though his attitude was kindly and his sense of humour keen, his judgment of people was extremely critical but at the same time very sound and he was utterly intolerant of humbug in any form.

So completely was his life wrapped up in surgery and surgeons that he appraised not only a hospital but a city or even a country, by the work its surgeons accomplished. To him there was little else of interest.

He received his early education at Gordon's College and later obtained an M.A. degree with honours at Aberdeen University. He took his medical degree in 1903 from Middlesex Hospital, London, and later joined its surgical staff. It was in this hospital, where he had spent so much of his life, that he finally died on 5th September, 1960.

His early study of the classics gave him continuing pleasure and not only did he delight in quoting them in his writings but when the occasion called for it, would deliver an entire address in Latin. In his later years he was chairman of the Horatian Society of London.

Gordon-Taylor travelled widely; at first it was his vacations that he spent visiting surgical clinics on the continent of Europe and later his eloquent and forcible speaking as well as his surgical fame, drew invitations from many parts of the world.

He came to Australia in 1934 to conduct the Primary examination for the Fellowship of the Royal College of Surgeons and this was the first of many visits. It was on this occasion that he was made an Honorary F.R.A.C.S. In 1947 he delivered the Syme Oration of the Royal Australasian College of Surgeons and in that year was honoured by the University of Melbourne with an Honorary LL.D. It was with great pleasure that he accepted an invitation from the Alfred and St. Vincent's Hospitals in Melbourne to become an honorary consulting surgeon on their staffs. His keen appreciation of the work of the surgeons in this country gave us a warm place in his heart.

The honours conferred upon him in many countries by Universities and surgical Colleges and Associations were legion, as were the many famous lectures and addresses he was asked to deliver.

As an operator he was both bold and careful but he would be fearlessly radical if he judged that to be in the best interests of his patient. This attitude was exemplified in the hind-quarter amputation, an operation which he made peculiarly his own. The concentration and nervous energy which he gave to his work was probably only realized by his intimate associates. He never learnt to drive a car because he felt it would detract from this concentration but after a heavy operative session he would be driven far into the country until he had relaxed.

In this intense life of surgery he was greatly helped and supported by his charming and beloved wife and her death in 1949 was a tremendous blow to him.

He gave his services in both World Wars. In 1914-18 he served as a major in the R.A.M.C. and for a time acted as consulting surgeon to the Fourth Army in France. In 1939-45 he was appointed consulting surgeon to the Navy with the rank of surgeon rear-admiral and in that capacity served in many parts of the world.

For those of us who knew Gordon-Taylor and who had experienced his kindly interest and help, there will never be anybody quite like him. A large number of the present generation of surgeons in Australia owe him much for his encouragement and advice.

As a mark of appreciation and to perpetuate his memory, Australian winners of the Hallett Prize, given in connection with the Primary F.R.C.S. examination, in 1949 founded the Gordon-Taylor Prize to be awarded on the results of the Primary examination for the F.R.A.C.S. A further gesture of the esteem in which he was held by his colleagues on this side of the world, was the recent commissioning of a portrait of him by James Gunn, R.A., to be hung in the headquarters of the Royal Australasian College of Surgeons.

To those of his friends who were saddened and shocked by his sudden death, it is a consolation to know that in his 83rd year death came to him at a time when his interest in surgery and surgeons was as keen as ever and while he still felt that he was a contributor to the science and art to which he had devoted his life.

TOTAL BODY PERFUSION*

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THE use of total body perfusion to provide prolonged intracardiac exposure for the repair of cardiac defects is now routine surgical practice. It is the purpose of this paper to discuss our current concepts of the theoretical and practical considerations involved in the conduct of total body perfusion. The information referred to in the discussion is derived from experience with the first 50 patients submitted to open cardiectomy, using total body perfusion, at the Alfred Hospital.

Nomenclature

"Heart-lung by-pass" or "cardio-pulmonary by-pass" refers to the mechanical act wherein blood is conducted away from the heart through cannulae placed in the venae cavae, passed through an oxygenating apparatus, and returned to the systemic arterial tree. This arrangement reduces the blood flowing through the heart to the small volumes which flow from the coronary and bronchial systems, and enables intracardiac manipulations to be carried out under direct vision.

"Total body perfusion" refers to the total management of the patient during the period of heart-lung by-pass and includes, for example, the provision of adequate rates of blood flow and the control of the composition of the blood delivered to the patient by the pump-oxygenator system. Whereas heart-lung by-pass is an all or none affair, total body perfusion may be adequate or inadequate. The adequacy of total body perfusion is largely under our control and is undoubtedly a major factor in determining the ultimate survival of patients submitted to open cardiectomy using total body perfusion.

The complexity of the responsibility assumed when one submits a patient to total body perfusion is not apparent on superficial analysis. In the intact human the circulatory and respiratory functions are nicely geared to the metabolic needs of the patient. On the one hand these functions are grossly interdependent whilst, on the other hand, they

maintain a high degree of self-regulation. When one submits a patient to heart-lung by-pass a good deal of this intrinsic control is lost and it is the surgeon's unhappy lot to attempt to substitute extrinsic control over a large number of variables including rate of blood flow, arterial oxygen tension (pO_2), blood carbon dioxide tension (pCO_2), blood volume and body temperature. It should be clearly understood that the responsibility for the levels of these functions existing during total body perfusion and the degree of control exerted over them lies entirely with the surgeon. His attitude towards this problem must be quite arbitrary in the final analysis; the arbitrary attitude which we have adopted is that, wherever possible, we should try to imitate conditions existing before total body perfusion was commenced in the subject undergoing controlled respiration anaesthesia for thoracotomy. Data relating to these "normal" values is still incomplete but with a clear idea based on the available information, it is a very humbling experience to see how poorly we can approach this limited objective. In our experience the period of intracardiac exposure required for the correction of defects lies between ten and ninety minutes. Undoubtedly in the future, when attention is turned to the correction of more complex defects, longer periods will be required. Reports from other centres have indicated that successful results have followed operations involving ninety minutes of total body perfusion. However, reports of successful operations involving periods longer than this are not available and it is probable that between ninety and one hundred and twenty minutes is the limit of time consistent with survival after total body perfusion using present techniques. This fact underlines the imperfections of these techniques.

If the physiology of total perfusion is studied interrelationships between factors soon become apparent. Thus, although most would agree that a major factor in determining the choice of an optimal rate of perfusion is the oxygen consumption of the patient, the latter is to a large extent dependent on the former. Such circular situations are frequently encountered. The discussion of these

*Received for publication 8th March, 1960.

†This work was supported by a grant from the Life Insurance Medical Research Fund of Australia and New Zealand.

factors as separate entities is not meant to imply that they are independent functions.

In the discussion which follows we have elected wherever possible to express parameters in terms of the body surface area. Thus the perfusion rate is expressed as a "perfusion index", that is the rate of blood flow in litres per square metre of body surface area per minute. This unit is comparable with the better known cardiac index.

The oxygenated blood is returned to the patient by the de Bakey pump, the rollers of which are set so as to occlude a latex tube. The output of the pump is metered by a tachometer. Heat loss in the circuit is overcome by a heating element wound around the glass of the oxygenating cylinder. The temperature is continuously monitored by a thermistor placed in the end plate of the oxygenating cylinder.

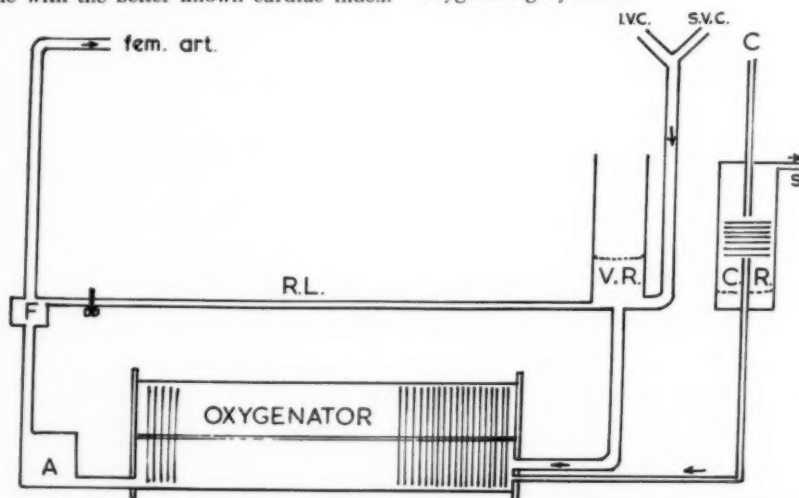


FIG. 1. The general arrangement of the pump-oxygenator apparatus. Venous blood is siphoned from the superior and inferior venae cavae (s.v.c. and i.v.c.) and collects in a venous reservoir (V.R.) which empties into a Kay-Cross spinning disc oxygenator. After oxygenation the blood is filtered (F) and returned to the patient's femoral artery by a pump (A). Blood is aspirated from the open heart (C) into a cardiotomy reservoir (C.R.) which is maintained at a negative pressure by suction (S). After deoxygenation this blood is also returned to the oxygenator. A recirculation circuit (R.L.) is occluded during total body perfusion.

MATERIAL, APPARATUS AND METHODS

Between March, 1957, and October, 1958, 24 perfusions were conducted using the Lillehei-De-Wall bubble diffusion oxygenator. Between November, 1958, and November, 1959, a further 26 perfusions were conducted using a modification of the Kay Cross spinning disc oxygenator. Most of the data referred to in the text and illustrations here were obtained in this latter period.

Venous blood is siphoned into a venous reservoir, the height and capacity of which can be altered to provide for maximum venous blood flow and to accommodate reserve volumes during perfusion (Fig. 1). The blood gravitates into the oxygenating cylinder where its level is maintained constant by manual alteration of the height of the venous reservoir. This allows for the most efficient use of the discs as oxygenating surfaces.

A recirculation line connecting the arterial line to the venous reservoir is used before by-pass is commenced and is occluded when the arterial line is opened at the beginning of by-pass. This provision facilitates heat conservation before by-pass and allows the blood to equilibrate with air, which is the ventilating gas used at this stage. When total body perfusion is commenced 95 per cent. oxygen and 5 per cent. carbon dioxide are ventilated through the cylinder at rates of flow greater than 10 litres per minute. The oxygenating capacity during perfusion is largely determined by the rate of rotation of the discs, which can be varied from 0 to 120 revolutions per minute. A Clarke polarograph electrode* inserted into the blood through the arterial end plate of the oxygenating

*Yellow Springs Instrument Company, Yellow Springs, Ohio, U.S.A.

cylinder continuously monitors the oxygen tension in the arterial blood leaving the machine. The operator adjusts the speed of rotation of the discs in accordance with polarograph readings, so as to attempt to maintain a level of arterial oxygen tension (pO_2) of 100 to 140 millimetres of mercury.

placement pump was measured by reference to a tachometer whilst, simultaneously, samples of arterial and venous blood were withdrawn for later estimation of the oxygen content by the Van Slyke technique. The results obtained are expressed in Figs. II, III and V. Whilst certain relationships are ap-

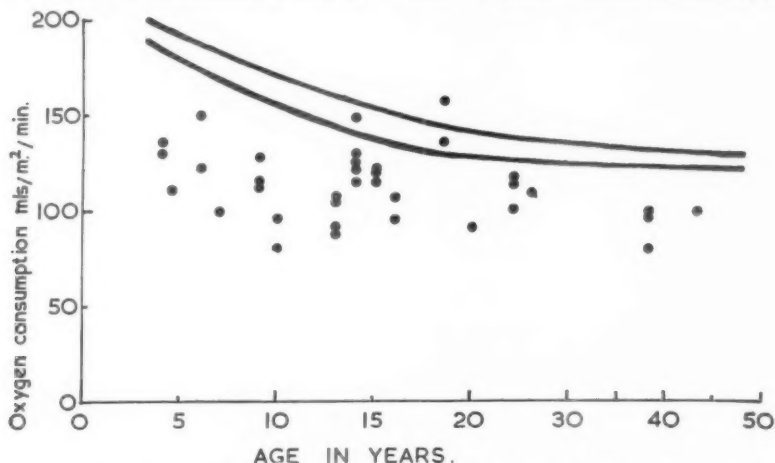


FIG. II. The effect of age on oxygen consumption during total body perfusion is expressed by the dots. The heavy lines plot the mean values derived from the data of Boothby and Sandiford relating the oxygen consumption of normal subjects under basal conditions. The upper line is for male subjects, the lower for female. The units used are millilitres of oxygen consumed per square metre of body surface area per minute.

The machine is constructed of glass, stainless steel and polyvinyl chloride tubing and can therefore be sterilized by autoclaving.

During perfusion samples of blood are taken from the arterial and venous lines for estimation of oxygen content, carbon dioxide content, pH and pCO_2 by standard laboratory methods.

The arterial and venous pressures are monitored by means of electromanometers connected to fine cannulae placed in the femoral artery and cephalic veins respectively. The electroencephalogram and electrocardiogram are also continuously monitored.

A. RESPIRATORY CONSIDERATIONS

1. Oxygen consumption during total body perfusion

The supply of an optimal amount of oxygen to the patient during total body perfusion must be regarded as of prime importance. If a measure of perfusion rate is available then oxygen consumption can be calculated by the Fick principle. The values for oxygen consumption presented here were obtained by this method. The output of a positive dis-

parent it is clear that there is a very considerable individual variation in oxygen consumption.

a. The effect of age

Boothby and Sandiford (1929) clearly showed that there was a progressive decrease in the total body oxygen consumption of normal subjects with advancing age. The rate of decline in oxygen consumption is small after the period of active growth and development has passed. A similar relationship has been found to hold during total body perfusion. Thus the mean value for subjects under the age of 5 years was found to be 126 millilitres of oxygen per minute per square metre of body surface area; whilst for those over 30 years of age it was 95 millilitres of oxygen per minute per square metre of body surface area (Table 1). Our experience does not include children under the age of 3 years in whom even higher rates of oxygen consumption are suspected. In Fig. II the oxygen consumption during total body perfusion is compared with solid lines derived from the data of Boothby and Sandiford. It is apparent that the oxygen consumption during total

body perfusion is usually about 25 per cent. less than that expected in the normal subject. The significance of this observation is discussed below.

surface area. In subjects over 1 square metre body surface area, the oxygen consumption was found to vary from 62 to 144 with a mean of 100 millilitres of oxygen per minute

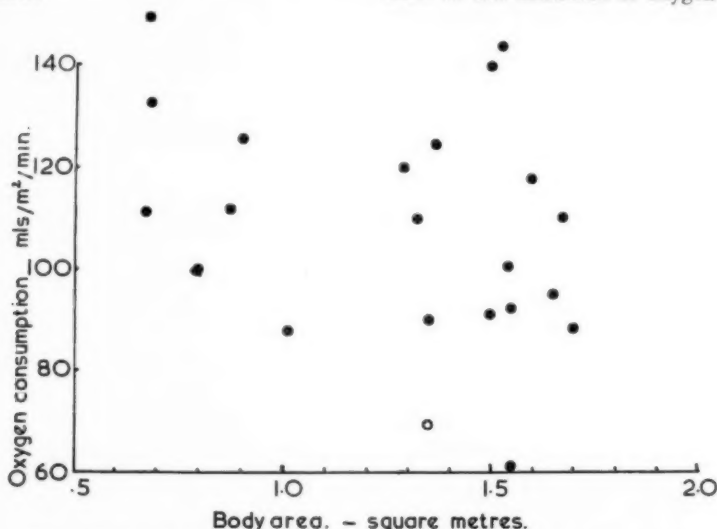


FIG. III. The relationship between the rate of oxygen consumption per unit area during total body perfusion and the body surface area. The oxygen consumption tends to be higher in small subjects but there is considerable individual variation.

b. Oxygen consumption and body size

The oxygen consumption measured during total body perfusion increases with increase in body size. In small subjects the oxygen consumption expressed per unit of body surface area is higher than in larger subjects (Fig. III). In subjects under 1 square metre

is pointed out that we have had no experience with the perfusion of subjects with a body surface area less than 0.5 square metres but it seems probable that the oxygen consumption of these small subjects would be higher and perhaps of the order of 150 millilitres of oxygen per square metre body surface area.

When the same data were analyzed in terms of body weight, it was found that the oxygen consumption varied from 6.5 millilitres of oxygen per kilogram per minute in small subjects to 2.4 millilitres of oxygen per kilogram per minute in the larger subjects.

c. The effect of body temperature

An abundance of literature attests to the effect of body temperature on oxygen consumption. As the temperature during total body perfusion has been rigidly controlled, information on this subject cannot be supplied from our experience with human perfusions. Fig. IV expresses the results obtained in a series of experiments on 9 dogs submitted to bi-ventricular by-pass and hypothermia by the technique described by Drew (1959). It is seen that there is a linear reduction in oxygen consumption with decreasing body temperature. A fall of 10°C. in body temperature

TABLE 1
AGE AND OXYGEN CONSUMPTION

Age	Mean O ₂ Cons ml/M ² /min.
0 - 5 years	126
5 - 9 "	121
10 - 14 "	110
15 - 19 "	112
20 - 30 "	106
30 - 40 "	95

body surface area, the oxygen consumption during perfusion was found to vary from 100 to 150, with a mean of 123 millilitres of oxygen per minute per square metre body per square metre body surface area. Again it

is associated with a reduction in total body oxygen consumption of 40 to 50 per cent. It is expected that similar relationships would hold during total body perfusion in human subjects. The body temperature during total body perfusion has been found to fall by about $2^{\circ}\text{C}.$, which might account for a 10 per cent. reduction from the expected oxygen consumption derived from normal subjects.

during perfusion increases with the perfusion index. It is more probable that at perfusion indices of less than 2.4 less than optimal amounts of oxygen are being transported to the patient's tissues or, in other words, some, if not all, areas of the body are going into a state of oxygen debt. Whether this effect arises because many areas are under-perfused, or because some areas are not perfused

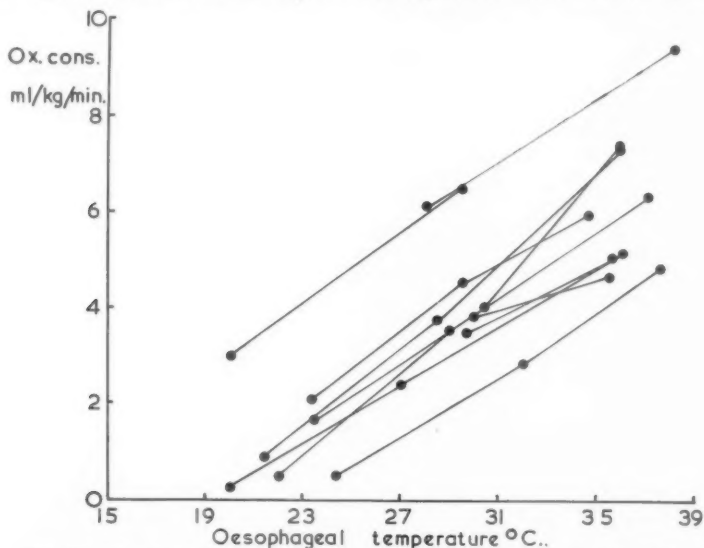


FIG. IV. The effect of body temperature on the rate of oxygen consumption. The graph summarizes the results of 10 experiments on dogs submitted to deep hypothermia and bi-ventricular by-pass. The rate of oxygen consumption (ml. per Kg. per minute) is plotted against the temperature measured in the oesophagus.

d. The effect of rate of perfusion

A number of investigators have shown that the oxygen consumption during total body perfusion is influenced by the rate of perfusion. Our experience expressed graphically in Fig. V, confirms this relationship.

In half of our patients the perfusion index was 1.5 to 2.4, the oxygen consumption during perfusion in these subjects varied from 62 to 177 with a mean of 100 millilitres of oxygen per minute per square metre of body surface area. In those patients with a perfusion index greater than 2.5 the oxygen consumption during perfusion varied from 100 to 150 with a mean of 128 millilitres of oxygen per minute per square metre body surface area.

It is unlikely that the metabolic needs of the patient during total body perfusion are governed by the perfusion index, although it is clearly shown that the oxygen consumption

at all, is a matter for serious investigation. Certainly at the practical level it is suggested that wherever possible a perfusion index of greater than 2.4 should be used to avoid a possibly dangerous situation.

While doubt still remains as to what constitutes optimal oxygen consumption during total body perfusion it has seemed reasonable to assume that the higher levels of consumption seen when higher perfusion indices were used, was an indication that the metabolic needs of the patient were being more adequately provided for under these conditions. It is also apparent that there is a very considerable individual variation which may be ± 30 per cent. of the expected mean for a particular patient. This fact renders prediction of perfusion factors difficult and points to the need for flexibility both in the capacity of the oxygenator and its mode of operation.

Within these limitations, the data obtained has proved useful to us in the planning of individual perfusions, since the capacity of our oxygenator can be varied within wide limits by altering the number of discs, their rate of rotation and the size of the oxygenating cylinder so as to suit each patient.

and practically possible to attain an arterial pO_2 of 600 mm. of mercury. The actual or potential danger of conducting total body perfusion under these circumstances is debated (Penido, Swan, Kirklin, 1957; Gollan, 1959) but in our view the evidence is sufficient (Kirklin, McGoon, Patrick, Theye, 1958) to

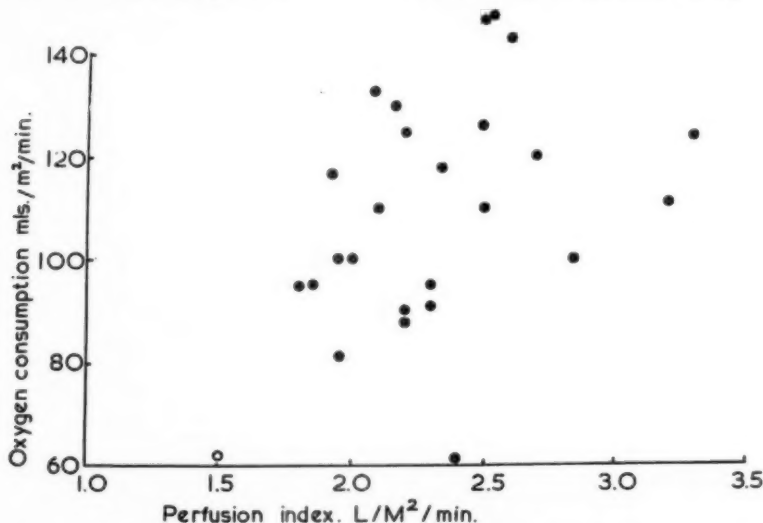


FIG. V. The relationship between the rate of oxygen consumption during total body perfusion and the perfusion index (litres per sq. metre body surface area per minute). Oxygen consumption increases with increase in perfusion index.

It is clear that with few exceptions, the oxygen consumption during total body perfusion is less than that expected in normal subjects under basal conditions. This reduction is probably accounted for by general anaesthesia, the lowered muscular work associated with controlled respiration, the decrease in cardiac work affected by cardiac by-pass and a moderate reduction in body temperature.

2. Arterial oxygen tension and saturation

It is within the capacity of most oxygenators to attain an oxygen saturation of 95 per cent. in the emergent blood. Few would dispute the necessity for attaining levels of saturation of this order. In the normal subject the arterial blood is 95 to 97 per cent. saturated with oxygen and its pO_2 is 100 mm. of mercury. In our view this is the optimal level during total body perfusion. It is not sufficiently appreciated that most oxygenating systems are capable of super-saturating the blood with oxygen. If 100 per cent. oxygen is used as the ventilating gas, it is theoretically

require steps to avoid this situation. By controlling the speed of disc rotation by reference to the readings obtained from the polarograph, it has proved a simple matter to maintain the arterial pO_2 between 100 and 180 mm. of mercury. Van Slyke estimations on blood sampled from the machine, confirm that the saturations of oxygen of the arterial blood have varied between 92 and 102 per cent. during perfusions in which polarographic control has been used.

Indirect evidence of the wisdom of controlling arterial oxygen tension has been observed in an improvement in the electroencephalographic pattern noted during these perfusions, in contrast to our earlier experience. Other factors may have played a part in this. Certainly there is good evidence from the work of Lambertson *et alii* (1953) that high tensions of oxygen in arterial blood cause an increase in cerebral vascular resistance and therefore, one would expect, a decrease in cerebral blood flow during total body perfusion if this factor were operating.

3. Venous oxygen saturation

The oxygen saturation of venous blood during total body perfusion is probably the best available single guide to the adequacy of oxygen transport. If the oxygen saturation of the arterial blood is fixed, then the oxygen saturation of venous blood is determined by the oxygen consumption and the perfusion rate. As we have already indicated, the

theoretical possibility still remains that, during total body perfusion, some areas may not be perfused at all and may therefore not contribute highly desaturated blood to the mixed venous pool.

4. Carbon dioxide transport and regulation

Adequate carbon dioxide elimination and the control of the tension of carbon dioxide in the arterial blood is an important aspect

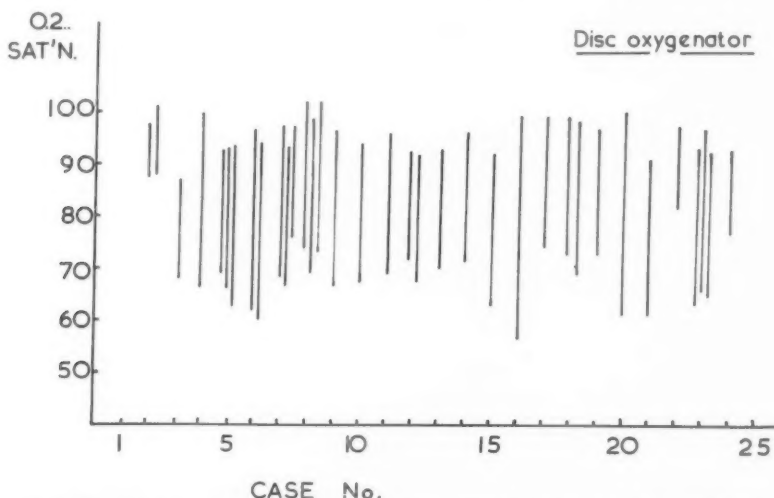


FIG. VI. Arterial and venous oxygen saturations measured during total body perfusion in 24 subjects. The straight lines join the points between the arterial and venous oxygen saturations and the length of the line is a measure of the arterio-venous difference. The mean venous oxygen saturation is 69 per cent.

former is subject to considerable variation, whilst the latter is largely under our control. A good case could be made out to control the rate of the arterial pump during total body perfusion, so as to keep the venous saturation between 70 and 75 per cent, that is within the normal range. This would overcome the problem of variation in oxygen consumption which, on the whole, cannot be predicted. In our experience the venous saturations during perfusion have varied between 58 and 88 per cent., with a mean of 69 per cent. (Fig. VI). It is clear that the perfusion index must be greater than 2.4 if an arterio-venous difference of less than 25 per cent. is to be consistently achieved (Fig. VII).

Whilst some comfort may be experienced by the surgeon if the venous saturation during perfusion is 70 per cent. or more, it must be realized that this only reflects a satisfactory state of oxygen transport if in fact all areas of the body are being perfused. The

of total body perfusion. The $p\text{CO}_2$ in normal arterial blood ranges from 40 to 45 mm. of mercury. During controlled respiration anaesthesia, levels of 20 to 30 mm. of mercury are frequently observed. The $p\text{CO}_2$ in arterial blood is a major determinant of cerebral blood flow and it has been established that hypocapnia results in decreased cerebral perfusion. Hypercapnia produces respiratory acidosis and is generally regarded as undesirable. The $p\text{CO}_2$ in the arterial blood leaving a pump oxygenator system is largely determined by the composition and rate of flow of the ventilating gases used. We have aimed at maintaining the $p\text{CO}_2$ between 30 and 40 mm. of mercury. The oxygenating cylinder is ventilated with a gas mixture containing 95 per cent. oxygen and 5 per cent. carbon dioxide at minute volumes of greater than 10 litres. The endogenous production of carbon dioxide by the body contributes so little to this great volume of gas that its effect is

inconspicuous. Results of determinations of the $p\text{CO}_2$ in the arterial blood in 18 consecutive cases, show that the mean value is 38 mm. of mercury and the range observed has been 30 to 44 mm. of mercury. We suspect that the provision of these tensions results in very little change in $p\text{CO}_2$ when total body perfusion is commenced and later discontinued.

5. pH of arterial blood during total body perfusion

Samples taken during total body perfusion indicate that the pH of arterial blood varied between 7.21 and 7.37. The mean value observed was 7.29. This value is rather lower

cedures but its presence in the context of total body perfusion should be presumed to be the result of anoxia until further elucidation of this problem has been achieved.

B. HAEMODYNAMIC CONSIDERATIONS

a. Hydrodynamic principles

The discussion of the respiratory factors relevant to total body perfusion leads us to the conclusion that the rate of blood flow through the pump oxygenator system should approximate to the resting cardiac output of the subject. If such rates of flow are to be attained, within the context of normal venous pressures in the great vessels, then very criti-

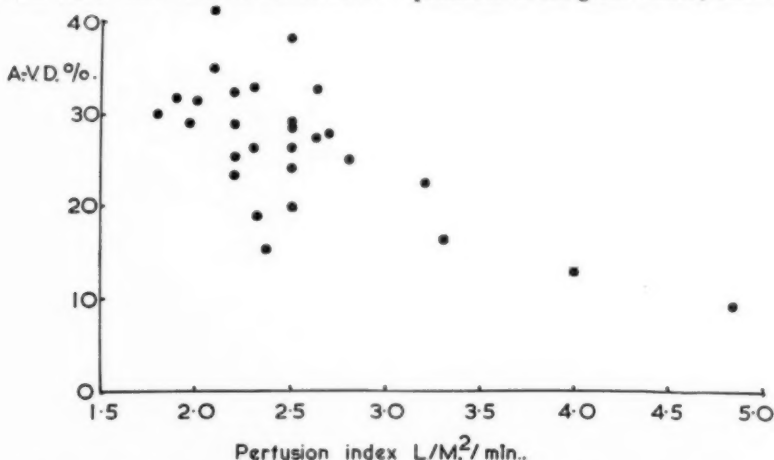


FIG. VII. The arterio-venous oxygen difference decreases with increase in the perfusion index. If the arterio-venous oxygen difference is to be less than 25 per cent., then a perfusion index of 2.4 or more must be used.

than that reported by other workers De Wall, Warden, Gott, Read, Varco, Lillehei (1956), and we suspect that it is lower because of the relatively high concentration of carbon dioxide which we have used in our ventilating gas mixture. It has been established that total body perfusion, even under ideal circumstances, does result in some metabolic acidosis and the pH of the arterial blood during perfusion will be influenced by this tendency and also by "respiratory" factors. Thus if one uses a low carbon dioxide tension in the ventilating gas there will be a tendency to respiratory alkalosis, with a resulting rise in the pH value. The low values of pH recorded during these perfusions, in which the carbon dioxide tension has been controlled at low normal levels, is indicative of the presence of metabolic acidosis. Metabolic acidosis is a frequent accompaniment of surgical pro-

cal consideration has to be given to the hydrodynamic design of the pump oxygenator and its conduits. The provision of adequate cannulation of the venae cavae and an appropriate artery is the most taxing technical feature of total body perfusion. The classical relationships enunciated by Poiseuille and others, relating to the laminar flow of homogeneous fluids through rigid tubes of constant diameter, bear little similarity to the conditions existing in the human body where a heterogeneous fluid flows through elastic tubes of variable diameter and flow is frequently turbulent. However these principles apply, to some extent, to the proper design of the extracorporeal circuit. The resistance to flow through a tube is proportional to its length and inversely proportional to the fourth power of its radius. Whilst it is important to reduce the length of all conduits to a minimum a

small increment in radius will effect a much greater reduction in the resistance to flow through it than a proportionate decrease in its length.

Attention must also be paid to the design of the whole of the extra-corporeal circuit so as to provide the maximum opportunity for laminar as opposed to turbulent flow.

of perfusion. It is helpful to distinguish between "total perfusion rate" and "the effective rate of systemic perfusion". Under the circumstances of cardio-pulmonary by-pass there is usually a small return of oxygenated blood to the left atrium, which presumably traverses the lung bed from the bronchial arteries. This detracts from the effective systemic perfusion

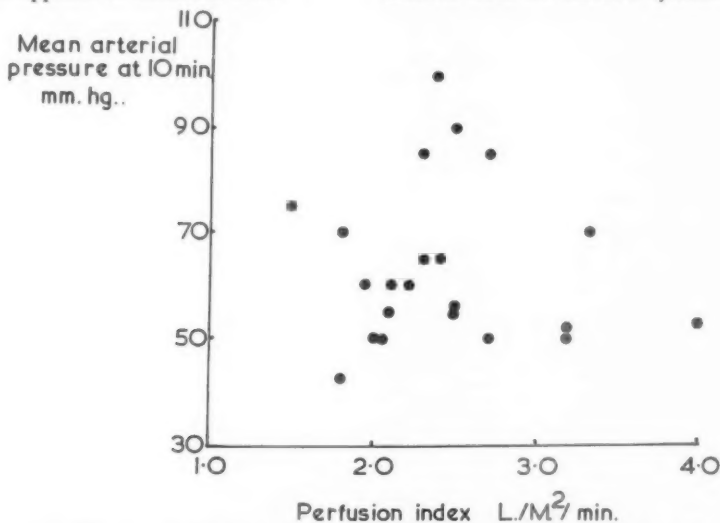


FIG. VIII. The relationship between mean arterial blood pressure, measured 10 minutes after the commencement of perfusion, and perfusion index. There is a very poor correlation between blood pressure and the rate of perfusion.

The factor of viscosity, which has a direct effect on resistance to flow, rarely enters physiological discussion since the haemoglobin content of blood is relatively constant. In the surgery of cyanotic heart disease however, great changes in viscosity may be encountered. For example, in one case of tetralogy of Fallot, the haemoglobin before operation was 26 grams per cent. During total body perfusion the haemoglobin content fell to 18 grams per cent., due to mixing with the donor blood in the oxygenator system. Four hours after operation the haemoglobin content was 15 grams per cent. The hypotension which follows operation on some cases of tetralogy of Fallot may be partly explained by this fall in viscosity which results from dilution due to mixing of polycythaemic with normal blood.

b. Perfusion rate

In the growth of knowledge of total body perfusion, few subjects have been as hotly debated as what constitutes an optimal rate

by a measurable but a relatively small amount. However, in some cases, for example, tetralogy of Fallot, where there are well developed bronchial vessels, the volume of this flow increases and may account for 30 to 40 per cent. of the total perfusion rate. This decreases the effective systemic perfusion considerably. The presence of aortic incompetence and the decreased resistance of the coronary vascular bed after induced cardiac asystole, may also result in significant falls in the effective rate of systemic perfusion.

If the postulates outlined earlier are accepted, then the optimal rate of perfusion is that which results in an arterio-venous difference of 20 to 25 per cent. In fact, this is found to require a perfusion index between 2.4 and 3. If there is little bronchial return then the effective rate of systemic perfusion is presumably also of this order. If, however, there is a large bronchial flow it may be necessary to use a total perfusion index of 3 to 4 to obtain an optimal rate of effective systemic perfusion.

The three factors which bear most directly on the choice of an optimal rate of perfusion from the haemodynamic point of view are the arterial blood pressure, the circulating blood volume and the venous blood pressure. These factors will be dealt with below. The question of venous pressure deserves special mention. The venous pressure during total body perfusion is to a large extent determined by the mechanical properties of the venous conduits, notably the resistance imposed by the venous cannulae and the venous lines, the pressure will be determined by the rate of flow through the cannulae and the resistance to flow through them; it may be physically impossible to attain high rates of flow if this cannulation is not adequate, so that, in practical terms, the situation that is frequently met is that the desirable rate of systemic perfusion cannot be obtained because of inadequacies of venous cannulation.

In the 50 cases submitted to total body perfusion at the Alfred Hospital, the perfusion indices have varied between 1.5 and 4.8, with a mean of 2.4. With our growing experience and reference to the experience of others, our ideas on arbitrary levels of perfusion rate have crystallized to some extent. Our present attitude is to attempt to attain a perfusion index between 2.2 and 3.4, in adults a figure of 2.2 has proved satisfactory, whereas in young children perfusion indices of 3.4 seem to be necessary to attain adequate venous oxygen saturations. In cases where the effective systemic perfusion is expected to be considerably lower, due to excessive bronchial flow, or aortic incompetence higher rates of flow than these mentioned are selected. A particular problem arises in small children with severe manifestations of the tetralogy of Fallot. The demand for high flows because of their age and because of the high bronchial flows may impose insuperable difficulties in the provision of adequate cannulation. A reasonable alternative approach to this problem is to combine hypothermia with total body perfusion and thus reduce the oxygen consumption, in this way total flows similar to those used in normal patients at normal temperatures, may suffice to supply an adequate effective systemic perfusion in this instance where the total body oxygen consumption is reduced.

c. Arterial blood pressure

Blood pressures recorded during total body perfusion when rates of perfusion approach

the normal cardiac index, are less than normal. This indicates a profound change in the factors included in the term systemic vascular resistance. In our experience, the blood pressure recorded 10 minutes after the beginning of total body perfusion has ranged from 43 to 100 mm. of mercury, with a mean value of 63 mm. of mercury. The mechanism of this is not clear but it may be due to disturbances of the reflexes, whose pressure-sensitive receptors are in the great veins and atria. There is a very poor, if any, correlation between the mean arterial blood pressure during perfusion and the rate of perfusion (Fig. VIII). This suggests that the major determinant of arterial blood pressure, during total body perfusion is the state of the systemic vascular tree.

The arterial blood pressure varies considerably during perfusions conducted at constant rates of perfusion. Commonly there is a fall in arterial pressure in the first two to three minutes of perfusion and this is followed by a fairly prompt rise to a relatively stable position after five to seven minutes. The subsequent changes in arterial blood pressure are unpredictable. In some cases there is a progressive rise in pressure with the passage of time whilst, in others, the arterial blood pressure remains fairly constant or may fall slightly.

The application of a clamp to the aorta, to induce cardiac asystole, usually results in a moderate rise in arterial pressure; its later release is usually associated with a significant fall. These changes are presumed to be due to changes in the effective rate of systemic perfusion, which results from preventing coronary blood flow, in the first instance and the excessive flow which occurs after myocardial anoxia in the second.

The lower-than-normal arterial blood pressures seen, even when there is a good effective rate of systemic perfusion, caused us some concern at first but they seem to be quite well tolerated. We have, on occasions, introduced nor-adrenalin into the arterial blood, which produced a marked rise in the arterial blood pressure but we are not convinced that this has increased the quality of the overall perfusion; indeed it may decrease the effective rate of systemic perfusion by causing an undesirable increase in coronary and bronchial blood flow. Until more evidence is available, it would seem wise to accept the low arterial pressures often seen during total

The perfusion blood volume is found to increase with increase in perfusion index. When the perfusion index is between 2.5 and 3 there is very little observable change in the patient's blood volume before, during and after perfusion. If low rates of perfusion are used, there may be very considerable decreases in perfusion blood volume during total body perfusion, which must be rectified immediately after the end of the period of heart-lung by-pass.

At any given perfusion index the perfusion blood volume is determined by the mean venous pressure, increasing if the venous pressure is high and decreasing if the venous pressure is low. The potential hazards of over-transfusion are well appreciated but little information is available concerning the effects of low venous pressure and low perfusion blood volume. Once more it is considered optimal to aim at achieving a normal blood volume by controlling venous pressure within the normal range.

Experiments using extracorporeal cooling indicate that the perfusion blood volume tends to increase with reduction in body temperature. This is the reverse of what one might expect from the vaso-constriction observed during surface cooling. The distribution of blood in the body under these circumstances is not yet defined. If total body perfusion is conducted in association with hypothermia this observation may be of practical significance.

Finally, in some conditions, notably those associated with a greatly enlarged heart for example in mitral incompetence, or a large left to right shunt for example in atrial septal defect, large volumes of blood which are usually contained in the heart or lungs may need to be stored during total body perfusion in the extracorporeal circuit. Provision for this should be made in the design of the pump-oxygenator system. Under these circumstances the maintenance of a "normal" perfusion blood volume in the patient could represent a state of significant hypervolaemia.

CONDUCT AND MONITORING OF TOTAL BODY PERFUSION

It is not intended to go into a detailed discussion of the conduct of total body perfusion but some reiteration of the principal features which we regard as important may be in order. We aim to deliver to the patient

body perfusion, conducted at rates of perfusion which would seem, from other criteria, to be adequate.

d. Venous blood pressure

While one might argue about the importance of monitoring arterial blood pressure during perfusion it is, in our opinion, imperative that the venous pressure is closely monitored—preferably in the drainage field of both venae cavae. The information thus obtained is necessary in the selection of the optimal rate of perfusion and in the detection of mechanical faults in venous cannulation, which might otherwise be undetected and therefore uncorrected.

The cannulae used must be thin walled, of large calibre and sufficiently rigid to avoid kinking, they must be placed in the cavae in such a way that they do not obstruct major venous tributaries. It is our practice to choose cannulae of a diameter equal to threequarters of that of the cavae. Since these necessarily impede venous return before and after bypass, the cannulation technique must be designed so as to allow them to be withdrawn rapidly into the right atrium if hypotension occurs.

The venous pressure recorded when a steady state is reached during perfusion is determined by the resistance of the whole venous conduit and the rate of blood flow through it. We have aimed to keep the venous pressure between 5 and 10 mm. of mercury and would regard a level of 15 mm. of mercury as the upper acceptable limit, beyond this it is probable that hypervolaemia and decreased tissue perfusion will occur. If the venous pressure is high and it cannot be lowered by attention to cannula placement, we elect to reduce the rate of perfusion.

A progressive rise in venous pressure to high levels during total body perfusion is indicative of obstruction to the venous cannula and is an urgent signal for the cause to be found and dealt with.

e. Perfusion blood volume

During total body perfusion the blood of the patient and the blood in the extracorporeal circuit freely mix. Observations on the volume of blood in the extracorporeal circuit give us a measure of changes in the blood volume in the patient; the term "perfusion blood volume" is used to indicate the volume in the patient and is a useful concept despite the absence of a notion of its absolute magnitude.

arterial blood at total flow rates sufficient to maintain an arterio-venous oxygen difference of 20 to 25 per cent. In our experience perfusion indices varying from 2.2, in the adult, to 3.4 in the small child are required. The arterial blood is ideally 95 per cent. saturated with oxygen at a partial pressure of 100 to 140 mm. of mercury, its $p\text{CO}_2$ is controlled between 30 and 40 mm. of mercury. Unless special indications are present for the use of hypothermia, total body perfusion is conducted at temperatures close to the normal body temperature. It is further aimed to keep the venous pressure below 10 mm. of mercury and the perfusion volume at an optimal level.

Total body perfusion is still a complicated procedure and it is highly desirable to reduce the use of monitoring devices to a minimum. In our opinion a reasonable compromise, between practical considerations and the desire to fully study every case, is to monitor venous pressure, arterial pressure, arterial oxygen tension, body temperature and the electrocardiogram. It is important to monitor venous pressure both for the selection of the optimal rate of flow and the detection of obstruction to the venous cannulae. It is debatable whether it is necessary to monitor arterial blood pressure during perfusion but there is no doubt of its value in the period immediately after heart-lung by-pass, particularly in the small child where cuff pressures may be difficult to obtain and unreliable. Those who believe that it is important to control arterial oxygen tension, as we do, will find the Clarke electrode a very simple and reliable piece of apparatus to use which does not add to the patient's burden, or significantly add to the pump operator's commitments. The electrocardiogram is not necessary for the conduct of perfusion but is occasionally valuable in the diagnosis of cardiac arrhythmias. In particular, in some cases of atrioventricular dissociation, the atrial rate may closely approximate to the ventricular rate, in these circumstances the recognition of this arrhythmia by inspection of the heart may be very difficult.

It has been our custom to monitor the electroencephalogram and by sampling techniques, the arterio-venous oxygen difference, the pH and the $p\text{CO}_2$. The information thus obtained has been invaluable in assessing the performance characteristics of our oxygenator system and in arriving at our present concepts of what constitutes adequate total body per-

fusion, but it is anticipated that we shall soon dispense with these at a time when a sufficient number of cases has been studied to complete our knowledge of these subjects.

SUMMARY AND CONCLUSIONS

Experience with 50 cases subjected to total body perfusion has been used as the basis of a discussion on the physiology of total body perfusion. Oxygen consumption during total body perfusion varies with age, body size, body temperature and the rate of perfusion. The significance of the reduced oxygen consumption during total body perfusion is discussed.

The arterial pressure during total body perfusion bears a poor relationship to the perfusion index. The significance of venous pressure and perfusion blood volume is discussed.

The problem of monitoring during total body perfusion is discussed. It is recommended that the minimum requirements for routine surgery include observations of the venous pressure, the arterial pressure, the body temperature, the electrocardiogram and the arterial oxygen tension.

ACKNOWLEDGEMENTS

The experimental development and later conduct of open heart operations using total body perfusion has been made possible by the generous co-operation of a large number of our medical, nursing and technical colleagues. It would be unjust to select individuals for separate comment.

We are grateful to Dr. T. E. Lowe for much helpful advice and criticism in the preparation of this paper.

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CAROTID BODY TUMOURS*

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THE tumours under discussion arise from the carotid body, which is an ovoid, pale-tan mass, rather poorly defined but measuring approximately 5 x 3 x 2 mm., situated a little medial and behind the bifurcation of the common carotid artery. It is quite firmly bound to these vessels by loose connective tissue and is probably best regarded as a specialized chemoreceptor which is affected by alteration in CO₂ and O₂ tension, by H⁺ ion concentration and by a range of drugs such as nicotine, cyanide and acetylcholine. It receives a fine nerve supply from at least the glossopharyngeal and vagus. The cause of tumour formation in these bodies is not known. Their histology has, however, been well described; the principal elements being the epithelial cell making up cell clusters together with scanty neural and endothelial elements. There is no evidence that these tumours arise from sympathetic nerve tissue or that they can secrete adrenaline: the terms paraglioma or chromaffinoma which are sometimes applied, are therefore inappropriate (Le Compte, 1943).

DIAGNOSIS

The importance of carotid body and allied tumours, such as paraganglioma intra-vagale, lies not in their frequency, for probably less than 300 have been described (Monro, 1950), but in the way a difficult decision concerning the life and death of the patient is suddenly thrust on the operator who comes unexpectedly on such a lesion. It is evident from a study of publications on such tumours that the majority are not diagnosed until exploration. One reason is surely that a carotid body tumour is so uncommon as to escape the differential diagnosis of a lump in the neck. Another explanation is the failure to elicit the physical characteristics of these tumours. Their site is characteristic, lying as they do in the crutch between the two major vessels bifurcating from the common carotid: the tumour, whose long axis lies in line with the

vessels, by its bulk and position bows the external carotid over its outer surface. This spread of the external carotid is readily ascertainable on palpation and is a characteristic produced by no other tumour. In consistency the tumour may be firm, or so compressible that its mass can be temporarily and markedly diminished by simple external compression. In the tougher tumours only transmitted pulsation will occur from the carotid wrap-around: the more vascular growths are both pulsatile and expansile. Application of a stethoscope over the tumour reveals a soft but readily detectable systolic bruit. In addition to lying in the plane of the vessels these lesions can quite typically be moved transversely but not axially. The vast majority of carotid body tumours are not tender to palpation and are pain-free, although in rare instances clinical manipulation or bending the neck in a tight collar may produce a syncopal attack. They have been described at any age from infancy onwards, although the majority are diagnosed in middle life. The sex incidence is approximately evenly distributed and the mean length of the history of the patient being aware of a lump is about seven years. On a few occasions (Monro, 1950), bilateral tumours have been described.

If, when faced with the differential diagnosis of a lump in the neck, the possibility of a carotid body tumour is both thought of and its physical features looked for, then there is little difficulty in making a probable diagnosis. In some cases because of the peculiarities of the tumour, the height of the carotid bifurcation, the size of the neck, or the scarring from previous exploration, the diagnosis may be less simple. In these patients we have found carotid angiography of considerable value. This not only reveals the tumour's outline but also the position of the arteries in relationship to the tumour, and most important of all, any large feeding vessels. The appearance of the carotid body tumour pushing out the carotid fork is most characteristic (Figs. I and II).

*Received for publication 25th March, 1960.

MANAGEMENT

The management of a carotid body tumour depends essentially on the predicted natural history of such lesions and on the morbidity or mortality accompanying its removal.



FIG. I. Case 3. R.E.M. Needle can be seen in common carotid artery. The internal carotid artery is well visualized and stretched around the tumour; the external carotid is mainly occupied in feeding the tumour, though two large branches can be seen arising from its lower end.

As the experience of any one individual or clinic is limited, answers to these questions must be made from published reports, remembering that the natural tendency is to publish the successful cases rather than the fatalities. With these limitations it is evident that while local recurrences following exploration and removal are not uncommon, distant metastases are rare (Le Compte, 1948). The latter do occasionally occur and there are numerous examples (Prendergrass and Kirsch, 1947; Donald and Crile, 1948; Turnbull, 1954; Hardy, 1959) where secondary deposits have passed to lungs and liver: secondary lesions in the spine may lead to paraplegia. Monro (1950) reported that "death directly attributable to the progressive growth of a carotid body tumour in cases either explored or abandoned, or treated by inadequate curettage or left entirely untreated, has occurred in 30%

of such cases". The natural history of these tumours then is such that the primary growth should be controlled unless age or other illness prohibits it. It would seem wrong to say, as some have done, that these tumours are essentially benign and slow-growing, therefore any attempt at operative removal, with its associated dangers, is contraindicated. On the other hand, in the group of published cases operated on the mortality is at least 15 per cent. If this is subdivided according to the procedure used, removal of the tumour leaving the carotid vessels intact and undis-



FIG. II. Case 1. J.B. Showing the "spread" of the carotid and the vascular nature of the tumour.

turbed has a 6.5 per cent. mortality; resection of the tumour with the carotid bifurcation or the simultaneous ligation of the common or internal carotid arteries has a mortality of over 30 per cent. Even so, Monro (1950) advises that if the tumour cannot be removed by dissection alone then it is advisable to resect the bifurcation. As none of the tests to predict the result of carotid ligation or the exercises designed to improve collateral flow are in any way reliable, we are unable to subscribe to this approach. Nor, in our limited experience of these tumours, has replacement of the internal carotid by a free graft seemed feasible. The difficult tumours are generally the large ones which reach high towards the base of the skull: resection of the neighbouring carotids only leaves a short stump at the cephalic end which tends to retract even more closely to the base of the skull following division. In the following case

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it was hoped that such a graft could, if necessary, be inserted: this was not possible, with a resulting fatality. This case also illustrates only too clearly the disaster of an unprepared exposure of such tumours not only immediately threatening the patient's life but also rendering further exploration very difficult.

Case 1

J.B., a female aged 13, was originally referred to a surgeon with a diagnosis of tuberculous glands in the left side of the neck. This mass had been observed for many months and as resolution was not thought to be occurring, it was decided that the gland mass should be excised. At the first operation the operator, immediately on incising the deep fascia and commencing dissection, was faced with severe bleeding which apparently arose from the mass itself: in attempting to control the bleeding the jugular vein was injured and the haemorrhage became more severe and difficult to control. Finally the common carotid artery was temporarily ligated (20 mins.), a transfusion of 6 pints of blood given and a pack introduced, which was afterwards removed.

The patient when referred to this clinic was seen to be a well developed girl of 13 with a painless mass 3 x 2 cm. located at the level of the upper border of the thyroid in line with the carotid artery. The mass would move laterally but not axially in the vessels, was faintly pulsatile and had, on auscultation, a soft systolic murmur. The diagnosis of a carotid body tumour was made and confirmed by angiography (Fig. II). It was decided to explore the mass because of the parents' anxiety over this, supported by the evidence that the tumour had on observation over the preceding few months been increasing in size.

At the second operation the tumour was found to be tightly adherent to the damaged jugular vein and could only be dissected off with great difficulty. It was further welded to the internal carotid artery by dense fibrous tissue, most of which probably resulted from the organisation of the previous haematoma. The external carotid artery, although wrapped round the tumour was readily cleared without damage. Additional exposure was obtained by division of the posterior belly of the digastric and the mobilization of the hypoglossal nerve. However, in removing the tumour at its uppermost point the internal carotid artery was so damaged as to be irreparable and as the line of section was virtually flush with the base of the skull, it was not thought possible to insert a graft or turn the external carotid into the divided proximal end of the internal vessel.

The patient never recovered consciousness, developed a profound R.hemiplegia and died five days later. Histological examination of the tumour revealed the characteristic picture of a chemodectoma with large cavernous blood spaces between groups of flattened parenchymal cells. There was nothing to suggest malignancy.

In the light of this experience our approach has been modified. If the tumour is readily resectable without division of the internal

carotid artery, this is carried out. As Gordon-Taylor (1940) has pointed out many of these tumours have a white line of dissection where they fuse with the arterial adventitia. Knife dissection in this layer is relatively bloodless as the tumour supply is largely derived from tiny arterioles rather than from large bore vessels. On two occasions during this above dissection the patient's blood pressure suddenly fell to below a systolic of 70 mm.Hg. Cessation of dissection with infiltration of the region with procaine brought this back to normal limits within a few minutes. This phenomenon we have attributed to a carotid sinus reflex. The following patient is an example of how dissection can be performed rapidly and with safety.



FIG. III. Case 2. W.S. Showing the encapsulated tumour with the clear line of dissection between it and the carotid vessel.

Case 2

V.S., a female aged 64, presented with a few months' history of a swelling in the left side of the neck. Her blood pressure was 160/100 mm. of mercury and she was otherwise asymptomatic. At operation a typical yellow-grey tumour, well encapsulated with a nodular surface and measuring 4.5 x 3.5 x 3 cm. was found in the carotid fork. The carotid bulb was infiltrated with local anaesthetic and the tumour readily dissected off from the artery to which it was bound by light connective tissue in which there lay small arteriolar-size vessels.

The patient made an uncomplicated recovery: histologically the tumour was a non-malignant chemodectoma containing cavernous spaces with a parenchyma of large polyhedral cells with granular, acidophilic cytoplasm and hyperchromatic nuclei. There was no evidence of malignancy.

There remains a group of patients in whom dissection with preservation of the carotids is not possible. This may be due to the scarring and destruction from previous exploration, the nature of the tumour in so much as it grows round the vessels or because of the high division of the common carotid (or in the rarer paraganglioma intra-vagale) the tumour lies high against the base of the skull only enabling the lower half to be exposed with safety. In these we feel, in view of the high mortality of carotid resection (at any age) and the dangers of vagal division (Bradbeer, 1959) that a compromise should obtain. The tumour is exposed between the carotid fork and dissected as free as is possible without danger to the major vessels. If there is a leash of vessels demonstrable at operation (see Fig. 1) or on angiography, these may be divided. With or without temporary control of the common carotid, according to the estimated vascularity of the tumour, its capsule, which is usually well defined, is then incised and by suction and morcellement the inside of the tumour is removed. Most of these lesions are so friable that suction alone is usually adequate. The centre of the tumour is then packed firmly with dry ribbon gauze until the bleeding is controlled and the capsule resutured. Following this the neck incision is closed and radiotherapy, to which these growths are moderately sensitive instituted (Doermann and Meffley, 1953). Only a long follow-up period will enable us to say whether this treatment will inhibit further recurrence: in view of the high mortality with bifurcation resection we suggest, however, that it is worth a trial.

Case 3

R.E.M., a male aged 51, had noticed a swelling under the angle of the L. jaw for eighteen months, which gave rise to no symptoms other than some difficulty on swallowing.

On examination the lesion was easily palpated, lying under the angle of the mandible and projecting some distance below. It was, however, more easily felt and seen by oral inspection, bulging into the oro-pharynx deep to the tonsillar bed. This mass was non-tender, had a light systolic bruit and was thought to be pulsatile. The patient had no evidence of a primary neoplastic lesion, no loss of hearing nor neurological signs.

This patient had been explored elsewhere with the reasonable diagnosis of a dumb-bell mixed parotid tumour in mind. It was evident when a total parotidectomy had unroofed the lesion that it was in fact arising from a deeper layer. An angiogram showed a large, well-circumscribed, highly vascular tumour displacing the left internal carotid artery

forwards and extending from the region of the carotid bifurcation to the base of the skull. At its lower tip a leash of vessels derived from the external carotid system could be seen to be entering. The diagnosis of a carotid body tumour or a paraganglion intravagale was made and the tumour re-explored.

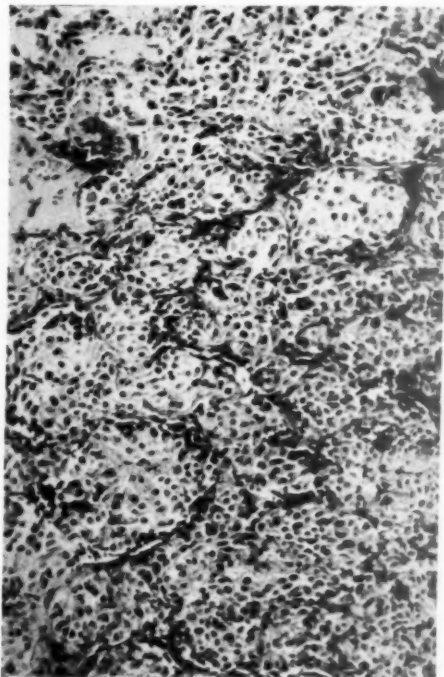


FIG. IV. Case 3. R.E.M. Typical cell pattern and organization of a carotid body tumour.

An incision anterior to the sternomastoid demonstrated the lower tip of the tumour around which the 12th nerve was coursing and from whose lower pole ran a number of abnormally thin veins: a leash of medium sized arteries entered the tumour from the external carotid artery at the same level. The tumour lay above and free from the bifurcation of the carotid. The tumour vessels were ligated, the posterior belly of digastric sectioned and the 12th nerve mobilized so that the lower two-thirds of the encapsuled mass could be exposed. It was not felt practicable or safe to dissect the whole tumour free: the capsule was incised and the centre of the tumour enucleated leaving only a thin shell lining the inner side of its containing membrane. The tumour was encephaloid and bled freely when removed by sucker and swab. The cavity was then packed with ribbon gauze and on cessation of the bleeding the pack removed and the capsule resutured.

The incision healed by primary union following which the patient received through a single lateral field (6 x 10 cm.) a dose of 6,400 r. (at 1 cm. depth) being administered in 32 doses over forty-six

days. The patient showed remarkably little pharyngeal reaction to this and at its termination the mass had subsided to a half its previous size.

Histologically the tumour consisted of epithelioid-like cells associated with connective tissue septa containing very numerous thin-walled vessels. In some areas the growth was intersected by fibrous tissue and hyalinized trabeculae. A few of the tumour cells were spindle shaped but the majority were polygonal with abundant eosinophil cytoplasm and indistinct cell outline. The nuclei which tended to be oval and vesicular showed some variation in size but without mitoses. Nerve fibres and ganglion cells were not demonstrated (Fig. IV).

CONCLUSIONS

1. The high mortality of carotid body tumours can be attributed to (a) the lack of a pre-operative diagnosis (b) the dangers of carotid bifurcation resection.

2. The clinical diagnosis of such tumours is discussed and the value of carotid angiography outlined.

3. It is suggested that it is never justifiable to resect the carotid bifurcation and instead tumours should be either removed by dissection with the preservation of the carotids or alternatively dealt with by intracapsular enucleation and radiotherapy.

4. Total justification for this policy must await long-term follow-up.

ACKNOWLEDGEMENTS

We wish to thank the various consultants who have referred these cases to us, to Dr. Begley for radiology and to the Institute of Medical and Veterinary Science for their histological reports.

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SHELLAC BEZOAR*

By J. P. AINSLIE

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THE development of a bezoar in the human stomach is not common. De Bakey and Ochsner comprehensively reviewed the literature in 1938, and collected 171 cases of trichobezoar, 119 cases of phytobezoar and 13 cases of other concretions, they added a further 8 cases of their own bringing the total number of recorded cases to 311. Haley, in 1957, was able to find recorded a total of over 400 cases in the world literature.

The trichobezoar is formed of human or animal hair, the phytobezoar of various forms of vegetable matter, those formed from per-simmon predominating.

Of the 13 cases of concretion 8 were composed of shellac, and occurred in furniture workers, who it is alleged drank furniture polish, a solution of shellac in alcohol, because of its alcohol content. Shellac, insoluble in water and in hydrochloric acid, is precipitated in the stomach to form a concretion or bezoar.

Inlow in 1940 reported a case of a shellac bezoar and reviewed 10 other cases from the literature. Inlow's was the first case reported in the English literature, 8 having previously been reported in Germany and 2 in Norway.

The first recorded case of shellac bezoar was reported by Tidemand of Norway in 1865. His patient was a 28-year-old prisoner, a drunkard, who died from perforation of a gastric ulcer, the stomach contained a large shellac bezoar weighing 1265 gm.

A history of having drunk shellac solution was obtained in all the 11 cases reviewed by Inlow. Of these 11 cases 3 presented with ileus due to impaction of a bezoar in the ileum, 3 died from perforation of a gastric ulcer associated with a gastric bezoar and others had symptoms suggesting gastric ulceration.

Apart from the case reported by Inlow in 1940 all cases were reported prior to 1918,

and with increasing world prosperity it is probable that the cult of drinking furniture polish will die and shellac bezoars will become a museum curiosity.

The occurrence of the condition in Australia is a rarity and the condition was only diagnosed at operation in the case now reported.



FIG. 1. Barium meal showing two foreign bodies in the stomach.

In December, 1959, a man aged 35 years presented with a history of severe epigastric pain over a period of five years; shortly after the onset he had suffered from severe haematemesis and melaena and had been admitted to a repatriation hospital for investigation. Barium meal investigation had failed to reveal a cause for his symptoms and he had been treated as a peptic ulcer. Symptoms persisted, and in spite of intermittent relief from diet and alkaline powders, progressively increased in severity. One month previously he suffered a further melaena.

*Received for publication 26th February, 1960.

Examination revealed a thin but otherwise normal looking young man of healthy appearance. His abdomen was thin, tenderness was present on palpation of the epigastrium but no mass was palpable. A diagnosis of peptic ulcer was confidently made and he was referred for barium meal investigation. This disclosed a large gastric ulcer on the lesser curvature of the stomach and two large negative shadows within the lumen of the stomach (Fig. 1). The radiological appearance suggested the presence of two smooth foreign bodies. As his sex made a diagnosis of trichobezoar unlikely and he denied having eaten any of the substances usually associated with the formation of phytobezoars the nature of the foreign bodies was not recognized.

Operation revealed a large ulcer on the lesser curvature and two large foreign bodies freely mobile within the stomach. Palpation through the gastric wall suggested they were composed of smooth plastic material.

Partial gastrectomy was performed, the two foreign bodies having the appearance of polished ebony (Fig. II). They had obviously formed within the

The bezoars were submitted to Dr. D. Curnow for biochemical examination and I am indebted to him for this report.

The patient was once a french polisher by trade. This suggested that the substance might be shellac. The other possibility was that it might have been a residue of wattle gum, to which he was addicted as a child. This latter idea seemed unlikely, as the gum is readily soluble in water. It was further investigated by testing for the presence of a polysaccharide.

A small portion was powdered and boiled with 2N sulphuric acid. The resulting solution, when neutralised with sodium hydroxide, gave no reaction with Benedict's reagent. Gum acacia, hydrolysed in a similar manner, gave a strong reducing reaction.

The following comparisons were made with a selection of solvents:

<i>Solvent</i>	<i>Bezoar</i>	<i>Shellac</i>	<i>Gum Acacia</i>
Water: Cold	insoluble	insoluble	soluble
Hot	insoluble	insoluble	soluble
Ether	partly soluble (yellow solution)	partly soluble (yellow solution)	insoluble
Alcohol	partly soluble (yellow solution)	soluble (yellow solution)	insoluble
N. Hydrochloric acid	insoluble	insoluble	soluble
N. Sodium hydroxide	almost completely soluble (violet solution)	soluble (violet solution)	soluble (colourless)
Dilute borax solution	almost completely soluble (violet solution)	soluble (violet solution)	soluble (colourless)

stomach and had been moulded to conform to the greater curvature, the larger of the two presented a concave surface on which the smaller rested.

It was considered the foreign bodies had been formed within the stomach, but the nature and source of the foreign substance was obscure. The appearance suggested a resinous origin and enquiry revealed that six years previously he had worked as a french polisher. He strongly denied ever having swallowed shellac solution or eaten shellac and as he appeared a well-balanced young man this denial was accepted.

Further enquiry revealed that he had lived on a farm until the age of 12 years, and like many others brought up in the country had frequently chewed and swallowed wattle gum.

A violet colour, similar to that given by shellac, was given when the material was formed into a paste with zinc oxide and alcohol.

From these observations it is concluded that the material consists principally of shellac.

Following this report the patient was once again questioned, and once again denied ever having indulged in the pernicious habit of drinking shellac solution. In the recorded cases of shellac bezoar three presented as

cases of intestinal obstruction due to impaction of the bezoar in the ileum, and it is difficult to reason why a smooth foreign body should remain in the stomach unless some degree of pyloric obstruction is present. In the case reported by Inlow an abnormality of the stomach was present and in this case a degree of duodenal ileus was observed during the barium meal examination (Fig. III) and confirmed at operation. It is suggested that delay in gastric emptying was a factor in the retention of the bezoar within the stomach.

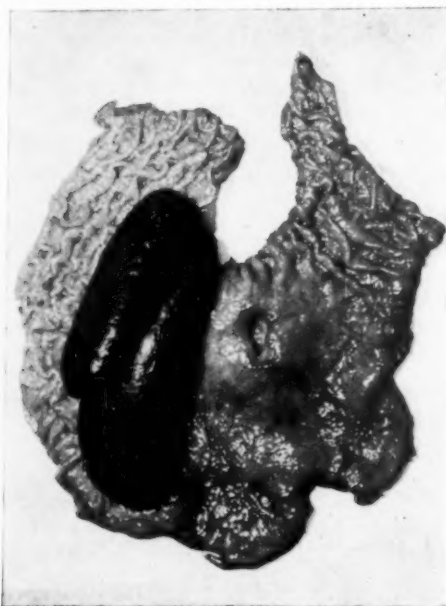


FIG. II. Portion of stomach removed showing a gastric ulcer on the lesser curvature and the two shellac bezoars.

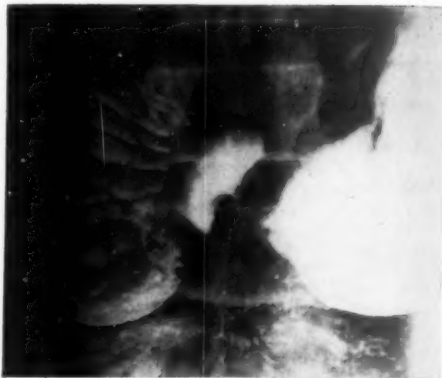


FIG. III. Barium meal showing duodenal ileus.

SUMMARY

(1) Eleven cases of shellac bezoars recorded in the literature are briefly reviewed.

(2) The condition occurs in furniture polishers addicted to drinking furniture polish for its alcohol content.

(3) A further case is recorded in a man who had previously been employed as a furniture polisher, but who denied having indulged in drinking the polish.

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THE APPLIED ANATOMY OF THE INGUINAL REGION*

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THE importance of the anatomy of the inguinal region lies particularly in its application to the surgery of inguinal hernia. It is not intended in this paper to discuss the incidence of this type of hernia, or the incidence of recurrence after operation. Suffice it to say that inguinal hernia is a common disorder, and that the results of surgical treatment, generally speaking, cannot be regarded as entirely satisfactory.

The problem of recurrence presents numerous facets, but even superficial enquiry reveals the importance of anatomy as a starting point. Reference to standard anatomical texts and original papers soon convinces the investigator that considerable difference of opinion exists as to what constitutes normality.

Methods of study commonly used include dissection at operation, at autopsy, and on the cadaver. Histological methods have been used by some investigators on adult and embryological material. Detailed observations concerning the controversial points present considerable difficulties. Studies made on the cadavers of elderly persons are limited by rigidity and atrophy of tissues, and by lack of definition of the various layers one from another. Observations made at operation are limited by time, by the presence of blood, and by restricted exposure. Studies should preferably be made of the anatomy in muscular young adult males soon after death and in a sufficient number of cases for the conclusions to be of statistical significance. However, such ideal conditions are practically impossible.

The author's interest in this subject was stimulated by experience with hernia surgery over almost twenty years, and by his performance of a number of groin dissections at autopsy in recent years. A technique to facilitate dissection has been developed subsequently which is, as far as he is aware, novel in this particular field of research.

MATERIALS AND METHODS

The observations recorded herein are the outcome of a series of dissections carried out by the author at autopsy on 25 male groins. In none of these was there evidence of a previous operation for hernia, but in a number of them the anatomy was complicated by the presence of a hernia of the direct type. In almost all cases the age at death was more than forty-five years.



FIG. 1. Left inguinal region. Infiltration deep to external oblique aponeurosis.

In addition, some dissections were carried out on females, but because the problems of hernia surgery in the female are similar to and secondary in importance to those in the male, no detailed recordings of these were made.

Dissection in all cases proceeded from superficial to deeper planes, commencing with a liberal skin incision. The external oblique

*Received for publication 15th February, 1960.

aponeurosis was divided from a point lateral to the anterior superior spine of the ilium to the pubic symphysis.

In order to determine the extent of origin of the internal oblique and transversus muscles from the inguinal ligament, coloured pins were used as markers for measurements.

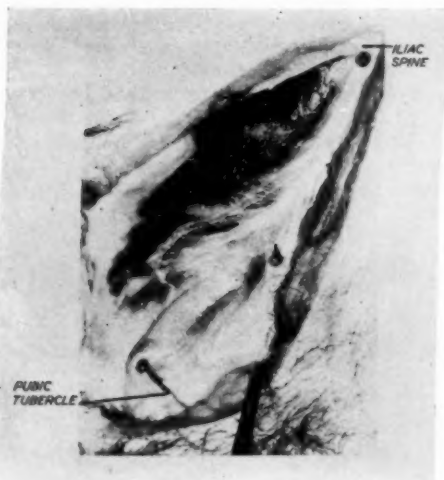


FIG. II. External oblique divided. Markers showing origin of internal oblique from inguinal ligament.

The difficulty of demarcating the various layers one from another, particularly where they are thin or adherent, was largely overcome by infiltration with coloured watery solutions. For this purpose a 20 ml. syringe and short-bevel 22-gauge needle were used. Infiltration was commenced at a site usually somewhat removed from the region to be displayed, where the plane between the two layers concerned could be located with certainty. Not only did this technique facilitate delineation of layers, but it also served to throw into relief the fibre arrangement of the more superficial layer, and to render its subsequent removal in the serial dissection easier and more complete. This applied not only to the layers of the abdominal wall proper, but also to their reflections on to the spermatic cord.

Features of the dissections were recorded photographically in colour and in black and white.

ANATOMICAL FINDINGS

External oblique (Fig. I)

This is entirely aponeurotic over the hernia region, and is generally a dense continuous layer, but in some instances it presents narrow longitudinal gaps over the inguinal canal. There is a considerable variability in the size of the so-called external "ring" (which is, in fact, triangular in shape) formed by the crura, and in the re-inforcement which this receives from the inter-crural fibres. Its lower edge, the inguinal ligament, is slightly convex towards the thigh and this convexity becomes



FIG. III. External oblique divided. Infiltration deep to internal oblique showing cremaster.

less evident after division of the deep fascia of the thigh, which is attached to the inguinal ligament throughout its length. This point has been made previously by Bracey (1956). As the dissection proceeds the inguinal ligament is seen in cross-section to be recurved on itself and the recurved portion expands slightly, flattening out as it proceeds, to its insertion into the pectineal line. These fibres constitute the pectineal portion of the inguinal ligament. The external oblique aponeurosis therefore is applied to the anterior, the inferior, and to a lesser degree to the posterior aspects of the spermatic cord. At the external ring this layer is continued as a covering of the cord into the scrotum.

Internal oblique (Fig. II)

This layer is musculo-aponeurotic. The variability of extent of its muscular origin from the inguinal ligament is evident from Table 1. In 24 measurements, 16 showed the origin to be between one third and one half of the ligament in its lateral portion. In 6 it was less than one third and in 2 more than one half. The proportion of muscle to aponeurosis in this layer is variable, as has been pointed out by Zimmerman and Anson (1953). Commonly it is muscular for one-third, and aponeurotic for two-thirds of its length from its origin to the linea alba. In one dissection the muscular fibres extended to within one inch of the linea alba. The aponeurotic portion fuses with the aponeurosis of the underlying

transversus muscle, usually a short distance lateral to the edge of the rectus muscle, but sometimes medial to this line. The muscular fibres of internal oblique describe an arch over the lateral part of the spermatic cord; then, becoming aponeurotic, the so-called "free" lower border fuses with the subjacent transversus aponeurosis and becomes the conjoined tendon, which in most cases is inserted into the pectineal line of the pubis. In some dissections no such insertion was present, and the conjoined aponeurosis merely found insertion via the anterior rectus sheath into the linea alba and pubic crest, as has been described by Zimmerman and Anson and others.

The cremaster muscle (Fig. III) is the continuation of the internal oblique on to the

TABLE 1
RESULTS OF 25 DISSECTIONS OF THE LEFT GROIN IN ADULT MALES AT AUTOPSY

Case No.	Age	Length of Inguinal Ligament	Origin of Internal Oblique	Ratio	Origin of Transversus	Ratio	Remarks
1	—	—	—	—	—	—	Trial of infiltration technique
2	C.45	11.4	3.8	0.33	3.8	0.33	
3	C.55	11.4	3.8	0.33	3.8	0.33	
4	63	12.7	7.6	0.60	3.2	0.25	
5	C.70	11.4	5.0	0.44	3.2	0.29	
6	60	12.7	6.3	0.50	2.5	0.19	
7	C.65	12.0	5.7	0.47	3.2	0.26	Potential indirect bulge
8	60	12.7	5.7	0.45	3.2	0.25	Direct hernia
9	58	12.7	6.3	0.50	5.0	0.40	
10	74	11.4	4.6	0.42	3.2	0.27	Direct hernia through conjoined aponeurosis
11	75	13.3	6.6	0.50	3.5	0.26	Direct hernia
12	79	13.0	6.3	0.49	3.8	0.30	Direct hernia with double sac
13	70	12.0	3.5	0.30	1.9	0.21	
14	47	13.3	6.0	0.45	4.1	0.30	Very muscular internal oblique
15	85	12.7	4.1	0.32	2.5	0.20	Direct hernia through conjoined aponeurosis
16	68	10.8	3.2	0.29	1.7	0.16	Direct hernia
17	64	12.0	3.2	0.26	3.2	0.26	
18	71	11.4	5.7	0.50	2.8	0.25	
19	85	10.8	3.2	0.30	1.7	0.16	Direct hernia
20	74	12.0	5.7	0.46	2.8	0.24	
21	72	12.7	6.3	0.50	3.2	0.25	Direct hernia
22	70	14.0	4.4	0.28	2.5	0.17	
23	84	12.0	4.4	0.37	4.4	0.37	Direct hernia
24	81	12.7	7.6	0.59	3.2	0.25	
25	25	12.0	5.0	0.41	3.2	0.27	

Measurements in centimetres.

spermatic cord. Its degree of development shows considerable variation in different bodies. Anteriorly its fibres arise from the muscular fibres of internal oblique as they arch over the cord. Inferiorly they arise from the deep surface of the inguinal ligament, down as far as the pubic tubercle. Superiorly they arise from the conjoined tendon down to the pectineal line and the pubic tubercle. These inferior and superior fibres are reflected on to the cord, and fan out over it to fuse with the fibres on its anterior aspect. The cremaster thus clothes the cord and testis. It is noteworthy that when the cord is lifted up from its bed, that is "mobilized" (as it frequently is in hernia surgery), to expose the posterior wall of the inguinal canal, the inferior and superior fibres of origin of the cremaster are necessarily severed (Fig. IV).

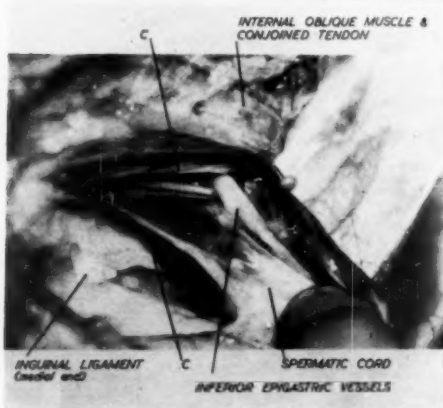


FIG. IV. External oblique divided, spermatic cord divided and mobilized, showing cremaster origin C medial to internal ring—see text.

Transversus

Just as with internal oblique, so also the origin of transversus from the lateral portion of inguinal ligament is variable in extent. Table 1 shows that in 4 out of the 24 measurements the origin was approximately one third; in 2 it was more than one third (but less than one half); in 18 it was less than one third, and in 12 it was less than one quarter.

The use of fluid infiltration left no doubt in the author's mind that the transversus muscle does contribute a layer to the cord coverings. In one of the dissections the contribution was fibro-muscular, but in others

it appeared more fibro-areolar to the naked eye (Figs. V and VI).

By further infiltration on the medial side of the internal ring it was apparent that this same plane of tissue forms the most anterior of the layers constituting the posterior wall of the inguinal canal. Furthermore, the fibres of this layer have a bipennate arrangement.



FIG. V. Internal oblique divided. Cremaster removed. Transversus exposed showing its extension on to spermatic cord.

The constituent fibres fill the elongated triangular gap between the conjoined tendon (from which they extend superiorly) and the pectineal portion of the inguinal ligament (from which they extend inferiorly). They converge and meet along a line joining the lateral end of the pectineal line and the centre of the spermatic cord at its point of entry into the inguinal canal (Fig. VIII). This layer varies, from one specimen to another, from being a firm aponeurosis with obviously tendinous fibres (Fig. VII) to a delicate membranous structure in which the fibres are little more than strands of areolar tissue. In the latter case the fibre arrangement described above may only be apparent when thrown

into relief by the subjacent coloured fluid used in infiltration or on close scrutiny (Fig. IX).

The fibres which stream off from the conjoined tendon are an expansion from transversus, and those arising from the inguinal



FIG. VI. Internal oblique divided. Cremaster removed. Transversus exposed with markers indicating its inguinal origin. Note extension of transversus on to cord and the presence of a direct hernia.

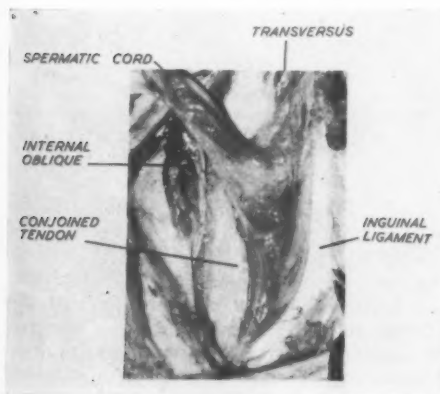


FIG. VII. Internal oblique divided. Cremaster removed. Cord divided and retracted upwards, showing bipennate fibres of transversus. Their origin from conjoined tendon and pectineal part of inguinal ligament, a suggestion of condensation of fibres on the medial side of the internal ring and reflection on to spermatic cord.

ligament should, in the author's view, be regarded as the continuation of the inguinal origin of the same muscle. Considered in this way, both internal oblique and transversus are seen to have a similar and systematic arrangement on the medial side of the internal ring. The most lateral point of convergence of the bipennate fibres, that is, where transversus expansion is reflected on to the back of the cord, is not always in close apposition to the cord, and there is, in these instances, a triangular defect in this layer, somewhat variable in size. The defect becomes apparent particularly when, in lifting up the cord from its bed, the fibres of transversus extending on to the medial aspect of the cord are divided. This point is demonstrated in Fig. X.

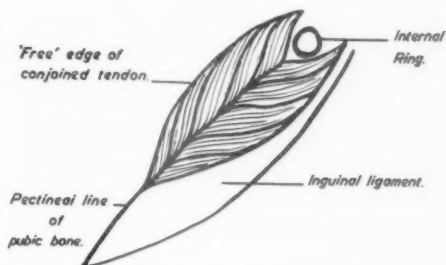


FIG. VIII. Showing the bipennate arrangement of the transverse aponeurosis behind the spermatic cord (diagrammatic).

Fascia transversalis

In the inguinal region, deep to transversus, lies a layer of tissue which is separated from the peritoneum by extraperitoneal fat. For purposes of description this is here referred to as fascia transversalis. It is a membranous sheet with condensations of fibres which are variable in their distribution and calibre, and lacking, in this series of dissections, any regular pattern. In particular, condensation around the cord at its point of entry into the inguinal canal was a feature in one dissection only, and was present in minor degree in two or three others. (These findings are at variance with those described by Lytle (1945). Lytle stated that a sling of fascia around the internal ring and attached to the deep surface of transversus was the usual finding.) The fascia transversalis was easily separable from the superjacent transversus after infiltration, except in the region of the bipennate fibres where close adhesion was commonly, but not invariably, found.

Where particular attention was directed towards determining the fate of the fascia transversalis in relationship to the cord an investment was demonstrable in some cases but not in others; when demonstrable it was a layer of very thin areolar tissue, sometimes complete, sometimes incomplete.

Along the line of the inguinal ligament there is continuity between the fascia transversalis and the fascia iliaca.



FIG IX. Magnified view of transversus medial to the cord, showing an example of fine fibro-areolar bipennate arrangement. (After removal of internal oblique, division and retraction of cord laterally.)

Extraperitoneal fat layer

This layer appears to vary in its fat content with the adiposity of the individual. In the same plane lie the bladder, the obliterated umbilical artery, the inferior epigastric and testicular vessels, and the vas deferens. The vas deferens and testicular vessels are, of course, the major central constituents of the spermatic cord, and the accompanying fat varies in quantity from being undetectable to the naked eye, to an amount constituting a lipoma of the cord.

Peritoneum

This constitutes a homogeneous layer which varies in thickness from one individual to another. In most cases, a small dimple was detectable at the internal ring, admitting no more than the tip of a probe; that is to say, the processus vaginalis was not patent. In one case the finger within the abdomen could be made to evaginate a small bulge of peritoneum through the internal ring. A hernial sac was

present in 9 of the 25 dissections, on the medial side of the inferior epigastric vessels. In 2 of these the herniation was through the conjoined tendon; in 7 it was in the more usual location of a direct hernia, that is, through the bipennate fibres of transversus.

Other workers have found a patent processus vaginalis in a proportion of groin dissections. Raw (quoted by Murray, 1906) found 9 instances in 200 dissections. Zimmerman and Anson (1953) found a patent processus in 20 instances in 100 dissections.



FIG. X. Internal oblique divided. Cremaster removed. Cord mobilized. Transversus exposed. Triangular defect in bipennate layer demonstrated. Small direct hernia more medially.

DISCUSSION

As has been mentioned, one of the major difficulties in this field is access to a sufficient number of satisfactory dissection specimens. The material available for the present work was far short of the optimum in that the number was small, the material was in many cases degenerate, and the picture was in some cases complicated by the presence of a direct hernia. In addition to the above the author, in agreement with Keith (1921), considers that the inguinal region in man must be regarded, relatively speaking, as a defective part of his anatomical make-up because of the common occurrence of hernia. Furthermore,

it is apparent that there is considerable individual variation in the local muscular topography.

Some of the points which the author wishes to emphasize, and which he has termed features, have been selected not so much for the frequency with which they have been observed in the dissections, but rather as they have appeared to represent an approximation to a state which may, for want of a better term, be referred to as "well-developed". Summation of those features gives a concept of what may perhaps then be referred to as the "perfect" state, rather than the "normal" state. It would seem that many groins are deficient in one or more of these features, and that the perfect groin is a rarity in the age group here considered. However, it is conceivable that further work on less senile material would do much to clarify this point. The author is ready to agree that in this concept of the perfect state he has been influenced to some extent by his views regarding the function of the inguinal musculature, to which further reference will be made in a later publication.

One might aptly describe the embryological descent of the testicle into the scrotum as a penetration of the abdominal wall. One would expect this organ to be clothed, as a result of its descent, by a representative of each of the various layers which it penetrates. The internal ring in the early stages of development lies more directly behind the external ring. The lateral shift of the former during growth to the adult state provides a rational explanation for the pattern of the reflections of the internal oblique and transversus on to the spermatic cord, on the medial side of the internal ring described above.

Before proceeding to the discussion of "the perfect groin" and its variations, some comments concerning controversial points are indicated.

Tobin *et alii* (1946) have discussed in detail the origin of fascia transversalis, and their paper is worthy of reference for those particularly interested in the derivation of this fascia. Briefly, the issues in this connection are whether the fascia transversalis should be regarded as a separate and individual layer, whether it consists of the epimysium on the deep surface of the transversus muscle, or whether, as Tobin and his co-workers have

chosen to describe it, the term should include the extraperitoneal fat as well as this membranous layer. The present author has used the term "fascia transversalis" to describe a distinct layer between transversus muscle and extraperitoneal fat.

The term internal spermatic fascia applies to one of the deeper investments of the cord. There is, however, no uniformity of opinion as to which layer of the abdominal wall this represents. Similarly there is lack of agreement about the part played by transversus and fascia transversalis with regard to cord coverings. Hayes (1950) regards the internal spermatic fascia as a continuation of the layer which he has named the abdomino-pelvic or parietal fascia, lining the whole abdominal cavity. Tobin *et alii* apply this term to an investment of the cord from what they regard as constituting fascia transversalis. According to Gray (1958) and to Hamilton (1956) transversus does not contribute to the coverings of the cord. Last (1954) and Grant (1958) describe the transversus and fascia transversalis as jointly forming the internal spermatic fascia.

In summary, the present work suggests that, in the perfect state the various layers of the abdominal wall in the groin exist as follows:

External oblique — a continuous strong aponeurosis with a small external "ring" reinforced by strong intercrural fibres, and without longitudinal defects.

Internal oblique—an origin from the lateral part of inguinal ligament approximating to half its length, a high muscle-aponeurosis ratio, and a well-formed conjoined tendon inserting into the pectineal line. It is noteworthy that such an arrangement provides for a relatively narrow triangular area behind the cord, between the conjoined tendon and the pectineal portion of inguinal ligament, whereas with a less extensive origin and an insertion of conjoined tendon not including pectineal line, the triangular area is relatively wide.

Cremaster — well-developed muscle fibres with a well-defined origin from the edges of the triangular area.

Transversus — an origin from at least the lateral one-third of inguinal ligament; a high muscle-aponeurosis ratio; a strong aponeurotic lower portion common to itself and

internal oblique (conjoined tendon); giving a fibro-muscular covering to the cord; finally presenting a well-developed bipennate arrangement, without a triangular defect on the medial side of internal ring, forming with the subjacent fascia transversalis a firm layer to which, in the author's opinion, the term "*bipennate barrier*" might appropriately be applied.

Fascia transversalis—a tough membranous sheet deep to transversus, having an extension on to the cord surrounding the vas, testicular vessels and a minimum of extraperitoneal fat, as a fine areolar layer. Some degree of condensation of fibres on the medial side of the inguinal ring is probably a part of the perfect state also.

Peritoneum—a uniform layer with no more than a dimple representing the processus vaginalis.

SUMMARY

Mention is made of the difficulties confronting the dissector in the inguinal region. A new adaptation of an old technique has been utilized to facilitate dissection of the groin in 25 adult males.

The findings are presented, and comparison is made with standard texts and other works, particularly in reference to the posterior wall of the inguinal canal and the coverings of the spermatic cord.

A concept is formulated, which is in fact a summation of the highlights of the dissections, and which is considered to represent the perfect anatomical state, although this may not necessarily be synonymous with the normal because of the wide variation in different

individuals, and because of the inherent deficiencies in this part of the anatomy of man.

ACKNOWLEDGEMENTS

The author wishes to thank the Superintendent of Prince Henry's Hospital and the Honorary Staff for permission to carry out this work. Thanks are due also to Dr. J. Funder and his staff in making the dissections possible. The advice and assistance of Mr. F. D. Burke and Mr. A. J. W. Ahern were of great help. Finally, the invaluable help of Mr. J. Scrimgeour on the photographic side, is acknowledged.

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INFECTION COMPLICATING HEAD INJURY*

THE VALUE OF PREVENTIVE SURGERY

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PYOGENIC infections of the central nervous system are still dangerous. While contemporary mortality rates for cerebral abscess, subdural empyema, and meningitis compare very favourably with those published thirty years ago, death from these diseases is still far too common for complacency to be justifiable. Pneumococcal meningitis for instance is often fatal, despite the extreme sensitivity of the pneumococcus to penicillin and other antibiotics. This is because the infection is commonly quite fulminating in onset. Staphylococcal meningitis again may be lethal, less from the virulence of the organisms than from their frequent immunity to antibiotics. The gram-negative bacilli can also cause intractable diffuse infections. Localized cerebral and extracerebral suppurations present similar problems. Although well-planned local and general treatment will save many lives, the residue of failures is too great to be ignored. Even in the best hands, the mortality from cerebral abscess is about 20 per cent. (Lewin, 1955).

It is therefore of great practical importance that wherever possible these intracranial infections should be prevented. Of the many causes of intracranial infection, trauma offers most scope for prophylactic measures. Our experience suggests that these opportunities are not always taken. It is our purpose to emphasize the importance of some simple surgical principles in preventing post-traumatic infection, whether early or late.

PATHOLOGY

Neural tissue has strong defences. The integuments of the brain and spinal cord (particularly the dura mater) are very resistant to external infection, but cranial trauma and much more rarely spinal trauma can breach these defences.

A penetrating wound may carry organisms into the substance of the brain. These will be

especially dangerous if foreign matter is also introduced; fragments of clothing, missiles and even bone chips greatly increase the likelihood of suppuration and also impede natural cure of established infections. Such infections are prevalent in war, even in such surgically well-managed wars as the Korean campaign (Wanamaker and Pulaski, 1958), but are unusual in Australian civil practice. However, cerebral abscess from in-driven foreign matter is still common in Europe (Blüml and Kraus, 1959) and it must be remembered that the infection may develop after a latent interval of many years.

Even if the dura mater is intact, infection of the scalp or skull may cause meningitis or cerebral abscess by spread along emissary veins. Infection by thrombophlebitis seems to have been very common in the past but indolent scalp wounds and cranial osteomyelitis are rare today and their cerebral complications, so well described by earlier surgeons, are not often seen.

Another and equally important cause of cerebral infection is cranial trauma involving the ear, nasal cavity, or paranasal air sinuses which may harbour organisms. The pneumococcus is often present in the healthy nasopharynx; haemophilus influenzae and the gram-negative cocci are even commoner commensals. The middle ear and the paranasal air sinuses are considered to be sterile under normal conditions but mild infection is common.

If a fracture of the skull base tears both the overlying meninges and the underlying nasal, paranasal or tympanic mucosa, then the stage is set for intracranial infection. A fistulous track from one of the three cranial fossae may result, permitting the cerebrospinal fluid to leak from nose or ear (Fig. I. a and b). Temporary reversal of the pressure gradient by increased intranasal pressure (as in nose-blowing) may force air or nasal mucus back along the fistula. If the patient

*Received for publication 13th April, 1960.

harbours a fairly virulent organism, such as the pneumococcus, a severe meningitis is likely. Less virulent organisms may, though this is rarer, give rise to local suppuration.

This dangerous sequence may take place at any interval after injury. The natural repair of these internally compound fractures is often defective. Several factors determine this inefficiency of the natural reparative processes. The bone over the paranasal air sinuses and in the cribriform fossa is extremely thin; if it is cracked, a flap of dura mater may be caught in the gap and bony union is then impossible. A sleeve of arachnoid may enter the gap and act as a conduit for cerebrospinal fluid from the large basal cisterns (Fig 1a).

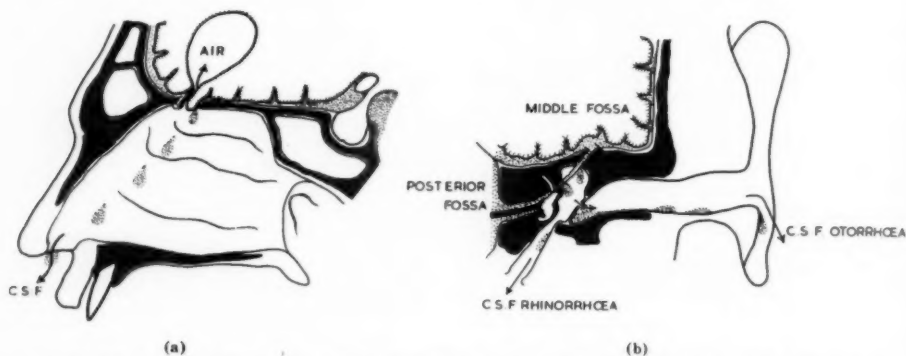


FIG. 1. (a) Diagrammatic sagittal section of anterior fossa of skull, to demonstrate C.S.F. rhinorrhoea and aerocoele complicating a fracture in the cribriform fossa.

(b) Diagrammatic section, obliquely through the petrous bone and ear, to demonstrate C.S.F. otorrhoea and rhinorrhoea complicating fractures either of the tegmen tympani or into the internal auditory meatus (alternative possibilities).

Cerebrospinal fluid rhinorrhoea may then continue indefinitely, as in some of the early reports reviewed by Dandy (1944). As long as the fluid leakage persists, the risk of infection is obvious, but even if, as more usually happens, the leakage ceases, the risk of infection is not abolished. Operative findings and autopsy studies (Linell and Robinson, 1941) have shown how flimsy the reparative tissues can be. Some cranio-nasal fistulae are closed only by plugs of brain tissue; the dural barrier is then not reconstituted and meningitis may flare up years after the original injury. One cannot of course say that efficient natural repair never occurs but it is certainly not to be relied on.

Fractures internally compound into the middle ear are less serious (Fig. 1b). The middle ear is usually sterile, and the Eustachian tube tends to prevent retrograde infection from the nose. Moreover, the thicker petrous bone is better able to reconstitute itself than the ethmoid, frontal and sphenoid bones. Nevertheless, the dangers of a fracture involving the middle ear, with cerebrospinal otorrhoea or other evidence of a cranio-aural fistula, are not negligible, especially if there is an associated otitis media.

MANAGEMENT

Open wounds of the head

In the last five years, the writers have treated 20 cases of cerebral or subdural abscess. In only one was the infection conse-

quent on an externally compound skull fracture. This case is, however, sufficiently instructive to justify a brief report.

Case 1

G.C., aged 45, fell from a truck and sustained a compound fracture of the right parietal bone. He was initially confused but responsive to command. He was admitted, after some delay, to a regional hospital, where the small scalp wound (about 2½ cms. in length) was probed but not excised or sutured.

His level of consciousness and general state deteriorated and four days after injury he was transferred to the Royal Adelaide Hospital. He was then comatose, responding feebly to painful stimuli. There was a left-sided hemiparesis and he had had several Jacksonian fits involving the left facial muscles and pharynx. His temperature was 101° F.

He was taken forthwith to the operating theatre, where a lumbar puncture was performed. The pressure was 120 mm. of water, the fluid was turbid and contained some 300 white cells (mainly polymorphonuclear), with 740 red cells and a protein content of 150 mgms/100 ml. The scalp wound was then excised. Under it, there was a circular depressed fracture, about 2.5 cms. across. Numerous hairs and in-driven dirt were seen in the edges of the fracture. The depressed bone was removed and pus began to trickle from a puncture in the dura mater. When this membrane was incised, a large subdural empyema was disclosed and evacuated. The patient made a slow recovery. Culture of the subdural pus grew a non-haemolytic staphylococcus. A more virulent organism might have been fatal. The patient has since suffered troublesome focal epilepsy not abolished by excision of a meningo-cerebral scar.

This unhappy story shows the importance of proper primary treatment of any head wound. The initial assessment, clinical and radiographic, must give as full a picture of the cranial and cerebral damage as is possible. If there is no skull fracture, the scalp wound can be sutured. It is essential that the scalp be shaved around the wound and that the wound itself should be cleansed of foreign matter. Formal excision of the wound edges can be omitted in small linear wounds but if there is much contusion or contamination, a marginal debridement is worth while. It is common practice to insufflate antibiotic powder but systemic chemotherapy is unnecessary and undesirable.

If the laceration overlies a fracture much more care is needed. A simple crack may be left undisturbed, provided that inspection shows no foreign matter between its edges. A compound depressed fracture however demands a considerable operation and the surgeon must be ready to manage haemorrhage or brain injury at short warning. Unless the depression is very slight (less than perhaps 5 mm.) it is elevated; if there is contaminated bone, it is nibbled away; if there is comminution, the devitalized fragments are removed. Unless the wound is very clean and very recent, we feel that the removal of comminuted bone should be generous as there is no great difficulty in secondary cranioplasty. An exception to this rule can be made for the supra-orbital region where the difficulties of perfect skull repair may justify the preservation of comminuted bone fragments, if still attached to pericranium and not overtly contaminated. Such conservatism is however a calculated risk.

If the dura is torn, there may be fragments of bone, or other extraneous matter, in the substance of the brain. Blunt violence, as in road injuries, rarely drives such debris very deeply but bullets and other missiles may penetrate to any depth. Contaminated foreign matter is a notorious source of infection and wherever possible these in-driven fragments should be removed. They may be seen in preliminary radiographs but such dangerous intruders as scalp tissue and pieces of hat will be invisible. If the cortex beneath the dural tear is lacerated, it should always be cautiously explored. Irrigation and very gentle suction will remove pulped brain and the track of a missile can be followed easily enough. Sometimes however, pursuit is unnecessary. Large, deeply situated missiles will sometimes retrace their path of entry under the influence of gravity, if the head is kept for a few hours in an appropriate posture. Soft bullets, fired point blank, as in suicides, tend to shatter and it is usually futile to pursue the multitudinous particles.

At the close of the exploration, the dura mater should if possible be sutured. This is always desirable and it is obligatory when the penetrating wound involves one of the paranasal air sinuses. If there is loss of dural substance, an immediate repair with pericranium or fascia lata can be carried out. However, this dural closure can at need be omitted, reliance then being on good scalp closure. If there is much urgency, or severe contamination, a free fascial graft to the dura may indeed be inappropriate (Lewin and Gibson, 1956). In any event, perfect scalp closure, without tension, must be secured. Full thickness scalp cover of the bone defect is essential; usually this can be obtained simply enough but if there is much loss of scalp, a flap should be rotated to cover the bone defect and the secondary exposure made good by skin graft on to pericranium.

In any compound skull fracture, prophylactic chemotherapy is justifiable. It is our practice to give penicillin and sulphadiazine in high dosage until the result of a preliminary wound culture is known. Usually these cultures are sterile but if a coagulase-positive staphylococcus is grown the appropriate antibiotic is given.

This policy of primary wound excision and closure, carried out as early as possible, is of course standard doctrine. But we feel that it is worth reiterating these well-known principles, because they are still frequently disregarded. If the surgeon is busy, or the patient critically ill, there is a strong temptation to cobble up the scalp wound and trust to kindly nature and a broad-spectrum antibiotic. While often such slovenly surgery succeeds, when it fails the forfeit can be dreadful.

In the last five years (to 25th Sept., 1959), the writers have carried out primary closure of 32 compound cranial fractures, including 6 due to missile injury. This is a small series, representing especially those cases where the severity of the injury, or some particular problem, caused the general surgeon to request neurosurgical aid. In 10 patients the accessory nasal sinuses were involved in the fracture. There were in the whole series only 2 examples of serious wound infection. Both resulted from disregard of the principles enunciated here. In one, the injury was a compound depressed fracture opening the superior longitudinal sinus, with much associated cerebral swelling. The patient was not expected to live and at the end of a tedious and difficult operation the scalp wound was sutured in haste and under some tension. It later broke down and a small cerebral fungus developed, with superficial infection. In the other case, the injury was a massive orbito-frontal depression. In an attempt to spare the supraorbital ridge and nasion, the surgeon did not remove several fragments of comminuted bone in those regions. This is always a calculated risk and is often successful, but, in this case, osteomyelitis and an extradural abscess followed. Sequestrectomy effected a cure.

Fractures involving the paranasal air sinuses and nasal cavity

These fractures may be considered as compound internally and in some instances they are also compound externally, with a wound of the overlying skin. They are well-known as a cause of intracranial infection and especially of meningitis. In a five-year period, 28 cases of pneumococcal meningitis were treated at the Royal Adelaide Hospital. In 7 of these, the infection was certainly consequent on injury to the anterior cranial fossa. Moreover,

of these 7 patients, 5 had had previous attacks of meningitis, one having had 4 separate bouts (Rischbieth, 1959).

Many of these episodes of meningitis could have been prevented by prophylactic surgery. This is demonstrated by a typical case.

Case 2

J.C., aged 50, was admitted to the Royal Adelaide Hospital, on 3 Dec., 1958, in coma. He was found to have severe pneumococcal meningitis, which responded to intraventricular and systemic penicillin. When he recovered, his history was pieced together.

In 1945 he had sustained a fracture of the skull, involving the anterior cranial fossa. As far as is known, there was no cerebrospinal fluid rhinorrhoea at that time.

In 1949 he had a severe attack of pneumococcal meningitis; when he recovered from this, he complained of a clear fluid discharge from the left nostril and of complete anosmia. He was investigated in a military hospital but no cranial fracture was demonstrated and nothing more was then done. The fluid discharge continued intermittently thereafter.

In 1954, he had a second episode of pneumococcal meningitis from which he recovered. The attack which was for us his presenting illness was the third in nine years.

These recurrent bouts of meningitis, associated with what was clearly cerebrospinal fluid rhinorrhoea, pointed to a post-traumatic cranio-nasal fistula.

Radiological examination of the orbital, frontal and ethmoid regions demonstrated a linear crack in the left cribriform plate. This side had been implicated by the history of rhinorrhoea. At operation (D.A.S.) the left anterior fossa was explored by a frontal osteoplastic craniotomy. A dural defect was found over the cribriform plate and here the frontal lobe was densely adherent to bare bone over an oval area about 15 mms. long. When these adhesions were divided, it was found that there was also a bony defect several millimetres across. A sheet of fascia lata was prepared and sutured over the whole area. The patient made a satisfactory recovery, and has been well (apart from headache) since.

Here and with 6 other patients, the first attack of meningitis could have been taken as a warning (Table 1). Repair of the anterior cranial fossa could have been done and this would almost certainly have prevented later attacks of meningitis. In several of these cases, the first attack of meningitis was preceded by cerebrospinal fluid rhinorrhoea. This should have given ample warning of the risk of intracranial infection.

It is true that on occasion the meningitis follows injury so closely that there is no time for preventive action. This however occurred only once in our series.

TABLE 1
MENINGITIS AFTER CRANIAL TRAUMA

Patient	Nature of injury	C.S.F. leakage	Number of attacks of meningitis	Interval after injury	Radiological findings	Treatment of bone defect
N.B. (N.S. Folio 878)	Ethmoidectomy	Rhinorrhoea for 18 months after injury	3 (2 pneumococcal)	1st: 18 months 2nd: 3 years 3rd: 8 years	Ethmoidal defect (seen in tomogram)	Fascial repair, recovery
J.C. (Case 2 in this report, N.S. Folio 991)	Closed frontal head injury	Rhinorrhoea, first noticed 6 years after injury	3 (2 pneumococcal)	1st: 4 years 2nd: 10 years 3rd: 14 years	Ethmoidal defect	Fascial repair, recovery
M.L. (Case 3) (N.S. Folio 871)	Frontal head injury with compound maxillary fracture	Rhinorrhoea in first 24 hours only	1 (pneumococcal)	24 hours	Aerocele; bone defect not demonstrable	Fascial repair, recovery
B.F. (Case 4) (N.S. Folio 1018)	Closed head injury (? site)	Otorrhoea from admission	1 (proteus vulgaris)	? 3 days	Aerocele and ventriculogram; fracture involving petrous bone	No operation, died
W.B. (N.S. Folio 728)	Closed frontal head injury	Rhinorrhoea, first noticed 14 years after injury	3 (pneumococcal)	1st: 10 years 2nd: 14 years 3rd: 15 years	Ethmoidal fracture (seen in tomogram)	Fascial repair; subsequent attack of meningitis; recent second repair with muscle graft
H.P. (N.S. Folio 3)	Compound frontal head injury	1. Alleged otorrhoea immediately after injury. 2. Rhinorrhoea noticed 5 months later	5 (2 staphylococcal) (2 pneumococcal) (1 unknown)	1st: 5 months 2nd: 11 months 3rd: 16 months 4th: 2 years 5th: 3 years	Frontal fracture; ethmoidal fracture not demonstrable	3 fascial repairs (one by another surgeon; eventual recovery)
D.S. (N.S. Folio 128)	Compound frontal head injury	—	1 (pneumococcal)	3 years	Frontal fracture	Fascial repair; removal of infected bone flap; recovery
R.P. (N.S. Folio 0523)	Compound frontal	—	2 (meningococcal)	1st: 7 days 2nd: 5 months	Bilateral frontal fractures	Fascial repair, recovery
P.Y. (N.S. Folio 0159)	Closed frontal head injury	Rhinorrhoea and otorrhoea from admission	2 (1 Friedlander's bacillus)	1st: 5 days 2nd: 3 weeks	No definite fracture	"Gelfoam" repair, recovery
L.J. (ACH Folio AC3116)	Compound frontal head injury	C.S.F. leakage noted during exploration of frontal sinus; no history of external leakage	3 (2 pneumococcal)	1st: 17 months 2nd: 20 months 3rd: 4 years	Frontal fracture, involving ethmoidal and ethmoidal sinuses	Fascial repair, recovery

Case 3

M.L., aged 44, was brutally injured in a fight on 7 June, 1958. He sustained a compound fracture of the nasal bones and maxilla, with backward displacement of the lower maxillary component. He was mentally confused when first admitted and it was considered impossible to immobilize his fracture. It was noted that cerebrospinal fluid was leaking from a laceration on the bridge of his nose. Radiographs of the skull, though taken under adverse conditions, nevertheless demonstrated a small subdural aerocoele in the frontal region.

The patient was given intramuscular penicillin and streptomycin. Despite these prophylactic measures, he developed fulminating pneumococcal meningitis twenty-four hours after his admission and seemed likely to die. There was gross toxæmia and evidence of a tentorial pressure cone, both pupils being fixed to light. Ventricular drainage was carried out and he was given penicillin, streptomycin and chloramphenicol, both systemic and into the ventricles.

He made an unexpected and dramatic recovery. The facial fracture was reduced and immobilized by an external crano-maxillary splint. When this could be removed, efforts were made to demonstrate by radiography a crack into the paranasal air sinuses. No definite fracture was seen, but the history made intervention obligatory.

At operation (D.A.S.) two small pits were found in the cribriform fossa. There were cerebral adhesions to these pits and their dural covering was deficient, the bone being bare to an exploring probe. A fascial graft was therefore sutured over the cribriform fossa. The patient remains well, one year after his original misadventure.

In the last five years, 11 operations of this kind have been carried out on 9 patients who have had meningitis after trauma to the anterior cranial fossa (Table 1). In every case, a reasonably adequate cause for the meningitis has been found. In 7, there was a crack in the region of the cribriform plate and in several of these a probe passed into nasopharynx or ethmoidal air cells. In one patient, separate fistulae into the frontal and ethmoidal air sinuses were discovered. Communication with the frontal air sinus was also demonstrated in one other patient. Operative closure of these fistulae was effected with little difficulty. In the majority, fascial grafts were used, being taken from the fascia lata or the temporalis fascia. Such grafts, carefully sutured in place, become nearly indistinguishable from true dura mater in a few weeks (Lewin, 1954) and constitute a good barrier to infection.

These operations for recurrent post-traumatic meningitis can be considered prophylactic, in that they avert the very real risk of another perhaps final bout of infection.

Can still more timely prophylaxis be achieved so that the likelihood of future meningitis is predicted and forestalled?

Fractures of the anterior cranial fossa are very common and post-traumatic meningitis is relatively rare. Operative repair of the anterior fossa is not a procedure to be lightly advised. Nevertheless clinical experience and consideration of the known pathology, show that there are certain fractures of the anterior cranial fossa which carry a predictably high risk of future intracranial infection; high enough to warrant routine prophylactic surgical repair. These are of course those fractures with fistulous communication between the brain or its meninges and the nasal cavity or paranasal air sinuses (cranionasal fistula).

It had been emphasized that natural cure of such fistulae is remarkably inefficient. It is therefore incumbent on the surgeon to carry out prophylactic repair of the damaged anterior fossa wherever there is certain, or strong presumptive, evidence of a fistulous communication, provided always that the patient is in a fit state for major surgery. Such evidence is given conclusively by the finding of cerebrospinal fluid rhinorrhoea, or by the demonstration of an intracranial aerocoele.

These conditions may demand operation in their own right. Profuse cerebrospinal fluid rhinorrhoea is a most annoying symptom and an aerocoele may function as a dangerous space-occupying lesion.

Our concern however is with the significance of these phenomena as warnings of future intracranial infection and in this context they are important even if evanescent or unremarked.

They indicate a grave and immediate risk of meningitis or cerebral abscess and this risk remains until the causative fistula is occluded. Even prophylactic antibiotics do not eliminate this risk, though they doubtless reduce it; two of our patients developed meningitis despite preventive therapy (Cases 3 and 4).

Cerebrospinal fluid rhinorrhoea

It is the practice in many neurosurgical units, including ours, to recommend prophylactic dural repair in nearly all cases where

this finding has been definitely established, even if the leakage has subsided (Lewin, 1954). Not all authorities accept this necessity. Rowe and Killey (1955) for example, contend that immobilization of a displaced maxilla will aid natural repair by protecting the cribriform plate from recurrent trauma (a proposition which all will accept), that penicillin and sulphadiazine provide reasonable safety while rhinorrhoea persists and that if the rhinorrhoea then subsides, no dural repair need be carried out. The latter propositions are not in accord with our experience. Dandy (1944) advocated dural repair only if the rhinorrhoea persists for more than two weeks. McKissock (1952) also has advocated conservative management. We believe that the risk of delayed meningitis always warrants dural repair although an exception is made if there is difficulty in demonstrating the exact site of the fistula. A fracture into the frontal air sinuses is usually seen in standard occipito-frontal radiographs. Fractures into the ethmoidal air cells or nasal cavity are on the contrary hard to identify. This is particularly the case when the bony defect is in the bottom of a deep cribriform fossa. Basal radiographs, tomograms and special oblique views (Johnson and Dutt, 1947) may aid but are often difficult to interpret. A fistula in this region may be suspected if there is unilateral anosmia and unilateral rhinorrhoea. Sphenoidal sinus rhinorrhoea on the contrary is usually a profuse bilateral discharge, often intermittent and sometimes associated with intact sense of smell (Lewin and Cairns, 1951). The radiological demonstration of sphenoidal fractures is comparatively easy and if there is a fistula, the lateral picture may show a fluid level in the sphenoidal sinus.

There are therefore certain diagnostic difficulties which may qualify the policy outlined above. If the rhinorrhoea has ceased and if a full radiographic investigation does not locate the site of the fistula, it may be wise to defer exploration. A complete exploration of the anterior fossa necessitates a bifrontal osteoplastic craniotomy and this is a major procedure, considerably more formidable than a unilateral frontal exposure. Sometimes a limited bilateral exploration is possible through a unilateral approach when the opposite cribriform plate may be seen by the use of a nasopharyngeal mirror. If the rhin-

orrhoea persists, it is needful before exploration to confirm that the leakage is cerebrospinal fluid and to exclude a source other than the anterior fossa.

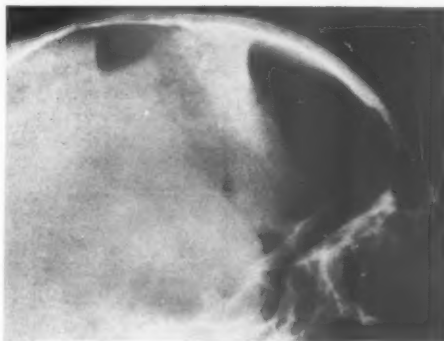


FIG. II. Traumatic aerocoele: air is seen, chiefly in the subdural space. This resulted from a fracture of the posterior fossa, entering the middle ear, and was associated with fatal meningitis (Case 4).

The fluid discharged in vasomotor rhinorrhoea (allergic rhinitis) can be profuse and watery. It can easily be taken for cerebrospinal fluid, especially as it may contain detectable amounts of glucose. It is however usually associated with nasal discomfort and contains eosinophil cells and nasal mucus. When the discharge is proven cerebrospinal fluid, it is only necessary to remember that rarely a fistula into the middle ear may present as rhinorrhoea, because the fluid is led into the nasopharynx along the Eustachian tube.

Aerocoele

Air may be insufflated through a defect in the anterior fossa into the subdural (Fig. II) or subarachnoid spaces, or even into the substance of the brain (Fig. III). It may enter the ventricles, achieving a traumatic ventriculogram (Fig. III). We contend that the finding of air within the meningeal envelopes has exactly the same significance as cerebrospinal fluid rhinorrhoea. In Case 3 subdural air was demonstrable in pictures taken a few hours before the onset of meningitis. Aerocoele may be suspected in a patient with an anterior fossa fracture who complains of persistent or delayed severe headache. The initial radiographs may not show the aerocoele, which can develop at an interval after

the injury, as in Fig. III which was taken 5 days after admission, the original pictures showing no intracranial air. Although some authorities (McKissock, 1952) dissent, we consider that the finding of an aerocoele warrants dural repair, provided that there is reasonable certainty as to the site of the fistula.

Are these the only indications for prophylactic repair of the anterior fossa?

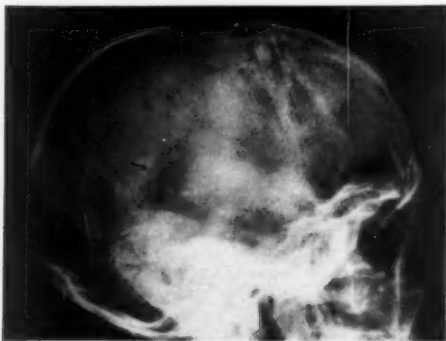


FIG. III. Traumatic aerocoele and spontaneous ventriculogram: air is shown in the frontal lobe and in the body of the lateral ventricle. These developed after a fracture of the anterior fossa, with an extensive dural laceration, which was repaired with fascial graft.

It has already been noted that externally compound fractures of the frontal region may involve the air sinuses and here a dural repair is certainly desirable. If it can be done at the primary operation, the ideal is achieved. If however brain swelling, contamination, or inadequate facilities prevent a satisfactory dural repair, it is wise to excise and close the external wound and to carry out formal repair of the anterior fossa after a suitable interval. In these patients the external violence has established a cranio-nasal fistula as well as an open wound and the long-term risk of intracranial infection is appreciable. A similar state is seen in some orbito-cranial wounds (Calvert, 1947), especially those resulting from suicidal attempts. The missile, entering through the mouth, the face, or the eye, may establish communication between brain and paranasal air sinuses and under some circumstances dural repair is proper.

Even in the absence of cerebrospinal fluid rhinorrhoea, aerocoele, or an external wound, there may sometimes be grounds for dural

repair. It is easy to overlook the occurrence of a small leakage of cerebrospinal fluid in the early days after a head injury. In the recumbent position, the fluid may trickle into the nasopharynx, and be swallowed. Moreover, in some fractures, the cerebral cortex is lacerated by spicules of bone and enters into direct relation with the open nasal sinus so that cerebrospinal fluid leakage may never occur. In at least three of our cases of delayed post-traumatic meningitis, there was no observation of initial rhinorrhoea, although the patients were in hospital and under careful observation for some time after their injuries. When there is radiological evidence of a sizeable defect in the walls of a paranasal sinus, or a fracture with marked separation of its edges, or a detached spicule of bone projecting into the lumen of a sinus and into the cranial cavity, then prophylactic exploration may sometimes be advised on the radiological evidence alone. Such cases are unusual and we have no example in this series.

It has been noted that, in the last five years, 11 operative repairs of the anterior fossa have been performed (on 9 patients) for prevention of recurrent meningitis (Table 1). In the same period 5 similar operations have been carried out for post-traumatic cerebrospinal rhinorrhoea and/or aerocoele. There have also been 10 cases of externally compound fractures involving the anterior fossa and in 6 of these formal dural repair with fascial graft was necessary either as a primary or a secondary procedure. There have thus been in all 26 operations which may be considered prophylactic against rhinogenic cerebral infection. Considering the group as a whole, the results have been satisfactory. There has been no death. There have been 2 cases of post-operative osteitis, one of the bone flap, the other of retained fragments of fractured bone. There has been one recurrence of rhinorrhoea and one attack of recurrent meningitis after such surgery and in these patients a second repair has been so far successful over periods of observation of four years and only two months respectively. In nearly every case, operation revealed the anticipated fistula. In one, exploration for presumed cerebrospinal fluid rhinorrhoea showed no intradural abnormality. In another the surgeon was sceptical of the significance of a defect with cerebral adhesions in the cribriform fossa. However, the defect was

covered with "gelfoam" and there has been no further rhinorrhoea. With these exceptions, the operations have seemed indubitably worthwhile, both from the conditions found at operation and from the post-operative progress. This experience accords with the reports of larger series.

Fractures involving the middle ear

These are quite common but usually carry little risk of intracranial infection. Bleeding from the external auditory meatus, or the observation of a haemotympanum, may indicate that the fracture involves the middle ear but a cranio-aural fistula is proven only by the issue of cerebrospinal fluid or the demonstration of an aerocoele. In the absence of either of these complications, conservative management is obviously sufficient.

Cerebrospinal fluid otorrhoea is usually seen when there is a fracture of the petrous temporal bone, tears of both the meninges and the mucosa of the tympanic or mastoid cavities and a defect (new or old) in the ear drum (Fig. 1b). If the drum is intact, cerebrospinal fluid can only escape along the Eustachian tube to the nasopharynx. Very rarely, cerebrospinal fluid otorrhoea may result from a direct fistula into the external auditory meatus. In general, such cranio-aural fistulae bear a relatively good prognosis and it seems that natural repair is more efficient in the petrous temporal bone than in the paranasal regions. Most writers advise expectant treatment, with prophylactic antibiotics, for cerebrospinal fluid otorrhoea. So treated, most patients do well and post-traumatic meningitis is rare. It is however not unknown. Wadsworth (1957) mentions one example among nine cases of traumatic cerebrospinal fluid otorrhoea. Linell and Robinson (1941) report 2 fatal cases. Shortman and Smith (1958) describe an interesting case, complicated by electrolyte depletion from fluid loss in which a pretraumatic otitis media was suspected. Clearly, pre-existing aural infection will very greatly increase the risk of cerebral infection.

We have had one relevant and distressing experience.

Case 4

B.F., aged 48, was admitted to the Royal Adelaide Hospital on 16 Jan., 1959, having injured his head in falling from a truck. He exhibited a right facial paralysis, and there was a leakage of blood-stained cerebrospinal fluid from the right external auditory

meatus. There was no history of aural disease. He was given penicillin and sulphadiazine in large doses. Radiographs of the skull showed fractures of the right occipital and petrous bones. Air was present in the subdural space, subarachnoid cisterns and lateral ventricles. The volume of subdural air later increased (see Fig. II).

On the second day after his admission his temperature rose and remained high. Lumbar puncture revealed a severe meningitis in which the infecting organism was *proteus vulgaris*. Despite energetic antibiotic therapy, this unfortunate man died. An autopsy revealed a large dural tear in the posterior fossa, with a fracture entering the middle ear.

Few surgeons see sufficient cases of this kind to form an opinion on the indications for prophylactic surgical repair. Natural cure seems usual and delayed meningitis from a persistent cranio-aural fistula must be very rare. It is our present belief that dural repair is justifiable if the otorrhoea persists for more than two weeks or if there is evidence of co-existing suppurative otitis, or if there has already been a bout of meningitis. It is to be remembered that exploration of the middle or posterior fossa is no small undertaking and it may be very hard to tell which region to explore. This difficulty is mentioned by Dandy (1944) and arose in several of the reported cases (Shortman and Smith, 1958). The middle fossa is relatively easily explored, although with rather more risk than the anterior fossa. Fistulae from the posterior fossa may issue into the internal auditory meatus, and be then undetectable. We have in one case explored both middle and posterior fossae, without finding a defect which happily has since cured itself. Ghouralal *et alii* (1956) demonstrated such a fistula by gravitating pantopaque from the cisterns of the posterior fossa into the ear; we have no experience of this method but intend to try it when occasion offers.

SUMMARY

1. The brain is well protected against external pyogenic infection. However its chief defence, the dura mater, has remarkably little capacity to reconstitute itself after injury. This is especially true where the dural injury results from fractures compound into the paranasal air sinuses. Reasons for this are discussed.
2. Infection complicating open wounds of the head can usually be prevented by proper primary wound excision, with removal of foreign matter and perfect scalp

closure. Dural repair is always desirable and it is essential if the wound also involves one of the paranasal air sinuses.

3. Infection, usually meningitis, complicating fractures involving the paranasal air sinuses and nasal cavity is common, dangerous and liable to occur at any interval after injury. If such an infection has occurred, repair of the associated dural tear is strongly recommended. Experiences in 10 such cases are presented.
4. Post-traumatic cerebrospinal fluid rhinorrhoea and intracranial aerocoele associated with frontal injury indicate a cranionasal fistula. Natural cure of such a fistula is unreliable and prophylactic surgical repair is advised. Exceptions to this policy are discussed.
5. Post-traumatic cerebrospinal fluid otorrhoea and intracranial aerocoele associated with temporal fracture present a more difficult problem and one less commonly encountered. The indications for surgical intervention are considered.

The substance of this report was delivered in an address to the South Australian Branch of the Royal Australasian College of Surgeons on April 15, 1959.

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THE TREATMENT OF PROLAPSED GANGRENOUS HAEMORRHOIDS*

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WHEN prolapsed haemorrhoids have remained unreduced for some time, gangrene, ulceration and sloughing are common sequelae (Fig. 1) and for these complications standard surgical opinion advises palliative treatment. This involves bed rest, usually with the foot of the bed raised on blocks (a procedure of doubtful value) and the application of compresses of perchloride of mercury, eusol or lead lotion. Gradual resolution follows over a period of two to three weeks, and although there is no doubt that the ultimate result will be satisfactory, a considerable period in hospital is needed for such palliative treatment. Thereafter, although a few cases will be found to have undergone spontaneous cure by reason of the sloughing which has occurred, the most common requirement is a further admission to hospital for haemorrhoidectomy. Immediate surgical treatment is usually rejected for two reasons:

- (i) the risk of spreading infection, and
- (ii) the risk of prolonged ulceration with stricture formation following.

The writer suggests that the further acceptance of this routine should be questioned. While in the minds of many there lurks the dread of precipitating an attack of suppurative pylephlebitis if prolapsed, thrombosed and infected piles are treated by operation instead of conservatively, actual investigation into the relationship of suppurative pylephlebitis with infected haemorrhoids reveals that such an association is extremely rare. Gabriel can recall no such case having occurred at St. Mark's Hospital, London, and Naunton Morgan can recall only one case, which occurred twenty years ago but the exact details of the condition of the haemorrhoids and their treatment are not available.

An extensive search of the literature reveals the report of only one other case of portal pyaemia which occurred in relation to haemorrhoids and this followed a routine haemorrhoidectomy. The patient recovered after a long illness in which subphrenic abscesses were drained (Alders, 1944).



FIG. 1. Prolapsed thrombosed and gangrenous haemorrhoids.

Lockhart-Mummery (1934) quoted a case in which a fatal result followed the conservative treatment of prolapsed gangrenous haemorrhoids. Gangrene of the rectum followed with extensive sloughing of the buttocks and finally, features very suggestive of portal pyaemia and septicemia. It is of

*Received for publication 26th April, 1960.

interest to note that in this case it was observed early in the history that "the veins of the rectum were thrombosed for two to three inches up the bowel."

Fatal complications of haemorrhoidectomy are fortunately very rare and of these, haemorrhage is undoubtedly the commonest. However, *Cl. Welchii* septicemia was the cause of death in a case of routine haemorrhoidectomy performed in Melbourne in 1957. The *Annual Reports of St. Mark's Hospital* (1928) record a death from gangrene of the rectum and pelvic peritonitis which

but it does not necessarily follow that a ligature and excision operation should be similarly censured. Although Gabriel (1945) states that "there is no more surgical reason for removing sloughing internal piles than there would be for doing a tonsilectomy for tonsillitis," the analogy is not a true one. Prolapsed gangrenous piles may in fact be removed as cleanly as may be a gangrenous appendix, without an incision being made through necrotic tissue. The writer's interest in this subject was stimulated by the observation that if a finger be gently inserted into the anus of such patients, the pile pedicles



FIG. II. When the haemorrhoids are drawn downwards, normal pile pedicles are seen.

followed an operation for prolapsed piles. This fatality of the pre-antibiotic era could possibly have been avoided by treatment now available. It may therefore be concluded that grave sepsis is an exceedingly unlikely complication of internal haemorrhoids and there is no evidence whatever to support the belief that, if it does occur in association with internal haemorrhoids, it is more likely to follow operative treatment. Indeed, it is even possible that the early, complete removal of the gangrenous tissues may lessen the risk of portal pyaemia and serious local spread of infection.

Many years ago it was customary to operate on such cases using the clamp and cautery method, an unanatomical procedure which rightly fell into disuse because of the likelihood of prolonged ulceration followed by subsequent stricture formation. All will agree with the condemnation of this method,

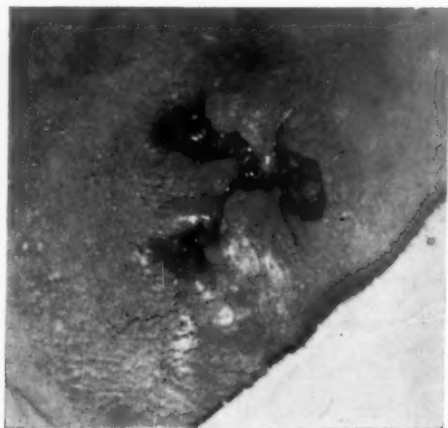


FIG. III. At the end of operation.

are usually found to be soft and unaffected; and, encouraged by this finding, operations have been performed on fourteen patients with strangulated, thrombosed and ulcerated haemorrhoids, including several where gangrenous tissue was present. When the pile has been drawn downwards (Fig. II), the usual scissor cuts, followed by horizontal dissection over the surface of the internal sphincter, will produce normal pile pedicles to be transfixed and ligated, as ordinarily, with strong linen thread or braided silk. Since the skin overlying the external haemorrhoidal plexus is distended and oedematous in these cases, allowance should be made for this by the provision of rather wider than usual "bridges," under which there may be thrombosed veins. Such veins may either be left to remain, in which case slow fibrosis

follows, or they may be gently filleted from beneath the bridges as a final step. The appearance at the end of operation in the case illustrated in Fig I is shown in Fig. III.

Many of these patients have had no bowel action for several days and it is wise to exclude the possibility of impacted faeces being present by a digital examination. Pre-operative manual removal may be required.

In all patients in this group treated by immediate operation, convalescence has been uneventful, differing in no way from other cases of haemorrhoidectomy. Antibiotics have been used, since their addition seems a reasonable precaution. It is therefore sug-

gested that since operation offers these patients a minimal period of hospitalization, with a saving of perhaps three weeks inability to work, its use should be seriously considered, especially when digital examination reveals normal pile pedicles.

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OBSERVATIONS ON THE SWOLLEN ARM AFTER RADICAL MASTECTOMY*†

By G. D. TRACY, T. S. REEVE, E. FITZSIMONS AND F. F. RUNDLE

THERE is a tendency to measure the success of the standard radical mastectomy for breast cancer only in terms of its control of the malignant process. Although this is the main consideration, the disability that may follow the operation must be borne in mind, particularly as other methods of treating breast cancer are available.

Swelling of the arm, frequently accompanied by discomfort, disfigurement and loss of function, is a common sequel which does not often receive mention in reporting the results of the radical operation. Very few reports include quantitative data on the extent of this problem. While accepted by most patients as part of the price of treatment for their cancer, arm swelling with its attendant pain becomes a source of misery to some, almost equal to that of the primary disease. In the Breast Investigation Clinic of this Unit, a quantitative study of arm swelling after radical mastectomy is being undertaken and this paper gives some of the results.

MATERIAL AND METHODS

A full clinical assessment has been made and the arm volume has been measured in a random sample of 106 patients previously submitted to radical mastectomy by a number of surgeons. The volume of both upper limbs and their segments are recorded routinely. The technique employed has been previously described in detail (Kettle, Rundle and Oddie, 1958). The measurements are made using the principle of water displacement (Fig. 1). The limb is immersed in a water-tank to a constant depth, the overflow being collected and measured. Successive readings show a high degree of reproducibility. Fore-arm (including hand) volumes are measured by immersion to the olecranon process and the upper limb by immersion to the axillary

fold. Arm volumes are given by the difference between the two.

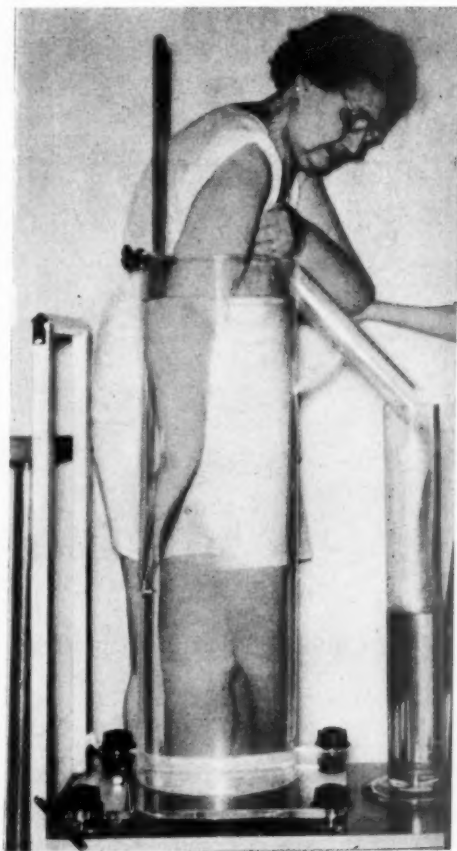


FIG. 1. Water displacement tank with depth scale for measuring arm volumes.

Additional observations, made on several patients, included phlebography, arm venous pressure recordings and visual lymphangiography after the method described by Kinmonth, Taylor and Harper (1955). Serial daily volume measurements were made on several patients receiving in-patient treatment for their arm swelling.

*Received for publication 28th April, 1960.

†This work was supported by a grant from the New South Wales State Cancer Council. Paper read by G. D. Tracy at the General Scientific Meeting of the Royal Australasian College of Surgeons, 20th August, 1959.

RESULTS

1. *Frequency of swelling*

Swelling of the limb on the same side as the mastectomy was evident clinically in 50 patients (47 per cent.), the swelling being classified arbitrarily as slight, moderate or severe as shown in Table 1. An additional 7 patients complained of swelling at other times but had none at the time of measurement. Five of these had had temporary swelling for a period of up to three years after operation but it had subsided spontaneously. One patient developed swelling after an injection of tetanus anti-serum in the arm. The ensuing oedema involved the whole arm and took several weeks to subside. Another developed post-operative venous thrombosis in the arm with severe swelling which gradually subsided during the next three years. In two, there was intermittent swelling which was not present at the time of measurement.

TABLE 1

INCIDENCE OF SWOLLEN ARM FOLLOWING
RADICAL MASTECTOMY
(106 cases)

	Number	Percentage
Insignificant (less than 150 ml.)	56	53.0
Slight (150-400 ml.)	20	18.8
Moderate (400-750 ml.)	17	16.0
Severe (over 750 ml.)	13	12.2
Total with swelling	50	47.0

It is established that many years may elapse between radical mastectomy and the onset of arm swelling. Since more than half of these 106 patients had undergone their operation less than four years previously, 47 per cent. probably understates the true frequency of this complication after radical mastectomy.

In this connection it may be relevant that 6 patients (5.6 per cent.) probably had swelling of the arm on the affected side. It was not evident clinically but the volume of the arm on the operated side was greater than its fellow of the opposite side by more than twice the standard deviation of the average

assymetry found in normal control subjects by Kettle, Rundle and Oddie (1958). Further observation is necessary to show whether such patients are more likely to develop clinical oedema.

2. *Onset*

In the majority of cases, the arm swelled early in the post-operative period. A considerable number observed that the swelling began immediately after X-ray therapy. In the minority, there was a latent period of up to five years in duration before the onset of swelling. Longer intervals were found in the series of 768 cases reported by Treves (1957). In our patients a variety of factors were noted in association with the late onset of swelling. These included trauma, inflammation, pregnancy, and the appearance of secondary malignancy. In one patient the onset of recurrent carcinoma produced bilateral swelling of the arms. Two patients with intermittent swelling noticed exacerbations before the menses, with improvement afterwards.

3. *Aetiology*

Phlebography frequently showed some constriction of the axillary vein with dilated collateral vessels. Withdrawal venous pressure tracings also showed an increase in venous pressure distal to the axilla in several cases. However it must be emphasized that no clinical evidence of venous congestion was seen in any of the patients with arm swelling, such as visible collateral or distended veins and erythrocyanoid discoloration of the skin due to capillary congestion.

During the period of this study we have had the opportunity of examining several patients with axillary vein thrombosis not associated with breast cancer. Although the presence of slight swelling may be established by measurement in these patients, it was never great in degree. Even when apparent clinically, the affected part pitted easily on pressure, and the limb showed the characteristic colour change and collateral dilatation of venous obstruction.

By contrast in the severer degrees of post-mastectomy swelling, the limb was waxen in colour and its appearance resembled elephantiasis due to lymphoedema. Visual lymphangiography by intradermal injection of a dye, pontamine sky blue, confirmed the presence of dilated dermal lymphatics with widespread patches of dermal "back flow"

higher up the limb pathognomonic of lymphatic obstruction (Kinmonth *et alii*, 1955).

Insufficient data were available in this series to evaluate the role of operative technique, wound infection and other factors in the etiology of arm swelling. There is no doubt that numerous factors operating singly or together may precipitate lymphoedema. One patient in the series developed lymphoedema after a radical mastectomy had been done unnecessarily for a benign lesion. Two others developed lymphoedema due to secondary cancer before any treatment had been undertaken.

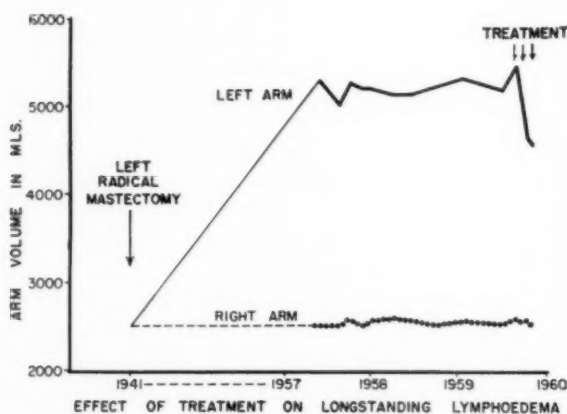


FIG. II.

4. Clinical features

Arm swelling usually involves the whole extremity. However in some cases the hand escaped and in others the forearm was not noticeably affected, the swelling being confined to the upper arm. In 2 patients only the forearm and hand were noticeably affected. Such unusual distribution is also occasionally observed in cases of primary lymphoedema of the lower limbs (Kinmonth, Taylor, Tracy and Marsh, 1957), in which swelling sometimes appears first in the thighs, although usually the ankle region is first affected.

In the early or slight degrees, the swelling may pit on pressure. In the gross degrees, it is hard and brawny. In such limbs lymphangiosarcoma may develop as a late and usually fatal complication (Stewart and Treves, 1948; Nelson and Morfit, 1956; Kettle, 1957).

Varying degrees of discomfort and loss of function were noted. Twenty-four of the 50 patients with swelling complained of pain in the arm. The arm was painful in only two of the patients without swelling. In some the pain was confined to the region of the shoulder joint, while in others it was more extensive; occasionally the whole arm ached. One patient had a classical thoracic inlet syndrome with pain radiating down the arm and acroparaesthesiae. Complaints of a feeling of tightness, heaviness and inability to carry loads with the arm were common. Two patients had to give up work and were virtually chronic invalids because of pain and swelling of the arm. Seven patients showed considerable limitation of shoulder joint movement.

In almost all cases the condition was made worse by excessive use of the arm and improved slightly on elevation and rest. Recurrent attacks of lymphangitis and cellulitis were noted in 8 patients (7.5 per cent.) of this series.

TREATMENT

In this communication nothing more will be done than to indicate certain possible lines of treatment. Care of the skin and avoidance of injury and infection are important in prophylaxis especially where measurements indicate that subclinical degrees of swelling exist. Several of our patients with established swelling gained symptomatic improvement from physiotherapy to increase the range of shoulder movement and massage to reduce the swelling. Serial daily limb volume measurements allow the efficacy of specific therapeutic measures to be followed quantitatively. Thus, one patient treated with a diuretic alone (chlorothiazide 500 mg. daily for four days) showed a reduction of 300 ml. in the volume of the swollen arm, with no effect on the normal arm. Another patient (Fig. II) was treated in hospital with chlorothiazide for four days together with elevation of the limb at night, using a pulley and counterweight. During the day the limb was massaged and compressed with an elastic bandage. In the course of five days the volume of the limb was reduced by 700 ml.

with considerable symptomatic relief. She was quite keen to arrange a similar suspension apparatus on her own bed and to continue treatment after her discharge from hospital.

Fitts *et alii* (1954) compared groups of patient with and without swelling with regard to 13 possible causal factors and found statistically significant differences ($P < 0.05$)

TABLE 2
INCIDENCE OF POST-MASTECTOMY LYMPHOEDEMA
(from literature)

Authors	Year	No. of cases	Incidence of swelling (percentage)
Holman, McSwain and Beal	1944	100	70
Nicolson and Grady	1948	230	44
Lobb and Harkins	1949	65	80
Daland	1950	90	55
Fitts, Keuhnelian, Ravdin and Schor	1954	130	64
McDonald and Osman	1955	143	72
Treves	1957	768	41
This series	1959	106	47

DISCUSSION

The frequency of post-mastectomy lymphoedema found in our series, namely 47 per cent., corresponds closely to the incidence reported in other studies which have given quantitative data (Table 2). It is known that relative deficiency in the lymphatics of a limb may exist for years without lymphoedema. Thus in primary lymphoedema swelling may only develop late in life when an unwonted fluid load is put on the lymphatics, as by injury, infections, or pregnancy. There seems no room for doubt that the arm swelling is, in fact, due to lymphatic oedema, despite the evidence for venous obstruction put forward by Veal (1938) and Parker *et alii* (1952). This evidence is based mainly on phlebographic findings and overlooks the absence of clinical signs of venous obstruction such as colour changes and dilated superficial veins. Halsted (1921) reported that excision of the axillary vein is rarely followed by a swollen arm, while MacDonald (1948) undertook routine resection of the axillary vein for stage 2 cancers and found a lower incidence of arm swelling in these patients than in those with conventional radical mastectomy.

in respect of three, namely, obesity, the number of nodes removed, and marginal necrosis of the skin flaps. There was less significant evidence that post-operative irradiation was a causal factor. However the association with irradiation, particularly given pre-operatively, was more strongly incriminated by Treves (1957). Irradiation was also thought to contribute to arm swelling by Neumann and Conway (1948) and MacDonald and Osman (1955).

The incidence of arm swelling with McWhirter's policy of radical radiotherapy without axillary dissection is not yet known. Kinmonth (1959), following a similar treatment programme, has noticed arm swelling only in patients with axillary metastases.

There seems little room for doubt that numerous factors play a part in the lymphatic destruction that results in post-operative lymphoedema and wound infection is generally agreed to be a major precipitating factor (Halsted, 1921; Holman, McSwain and Beal, 1944). Details in operative technique have been claimed to be important in preventing lymphoedema (Halsted, 1921; Daland, 1950). Measures to reduce the incidence of arm stiffness, fluid collections, flap

necrosis and wound infection by carefully planned incisions, skin grafts to prevent tension in closure and continuous suction drainage have been claimed by several authors (MacDonald, 1955; Brush *et alii*, 1958) materially to reduce the frequency of the swollen arm. It also seems logical to confine post-operative irradiation to the lymphatic areas not dealt with by operation, such as the internal mammary and supraclavicular nodes, when radical dissection of the axilla has already been carried out.

This study has shown that lymphoedema is not always progressive. Indeed, it occasionally subsides with the passage of time. Vigorous therapy is often effective in providing symptomatic relief. A combined programme of dehydration, sodium restriction and diuretics at the onset of treatment, accompanied by elevation at night, frequent massage of the limb and compression bandage or sleeve, should be undertaken in the patient with severe pain due to tightness of the arm (Foley, 1951). Ingenious machines have been devised to provide intermittent pneumatic compression while resting in bed and, for the patients who can afford this, the results are very gratifying (Brush *et alii*, 1958). Kinmonth (1959) has attempted a modified Sampson Handley operation to provide lymphatic drainage from the limb to the opposite axilla with fine polythene tubes. However only temporary benefit has resulted. He has also treated a few grossly lymphoedematous limbs by radical excision of the subcutaneous tissue, replacing the skin on the underlying muscle, as a free graft.

SUMMARY

The frequency of arm swelling after radical mastectomy has been studied in 106 cases using an accurate method of measuring arm volume (Kettle, Rundle and Oddie, 1958). Swelling was evident clinically in 50 patients (47 per cent.). Seven additional patients had temporary or intermittent swelling not present at the time of measurement. Six more had subclinical swelling when compared with the mean asymmetry of normal upper limbs.

Evidence is presented that the swelling is due to lymphatic oedema. Associated factors in aetiology are reviewed.

Post-mastectomy lymphoedema may cause considerable symptomatic distress and a positive treatment programme for these patients is advocated. The value of arm volume measurement in assessing the progress of the condition and the results of therapy is indicated.

ACKNOWLEDGEMENTS

We are grateful to members of the Honorary Surgical Staff of the Royal North Shore Hospital for permission to study some of their patients and to the General Medical Superintendent for permission to use hospital records.

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LYMPHANGIOMA-HAEMANGIOMA OF THE JEJUNUM: A RARE CAUSE OF ALIMENTARY TRACT BLEEDING*

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THE source of gastro-intestinal bleeding not infrequently presents a difficult diagnostic problem. In a recent discussion of alimentary bleeding of obscure origin, Avery Jones *et alii* (1959) estimates an overall incidence of 15-25 per cent. in all large series. Palmer (1952), in a thoroughly investigated series, reports an incidence of 14 per cent.



FIG. I. Surface view of operation specimen. The haemorrhagic polyp is seen projecting from the jejunal mucosa.

Rare causes are of importance when the source is not readily revealed by the standard tests including X-ray examination and when the lesion may escape the discerning eye of the surgeon when he finally performs a laparotomy. The following case of severe and continuing blood loss is reported here as the source of the bleeding, a mixed lymphangioma-haemangioma of the jejunum, nearly eluded all attempts at diagnosis.

*Received for publication 26th February, 1960.

†Pathologist to the Clinical Research Unit, working with the aid of a grant from the National Health and Medical Research Council of Australia.

CASE REPORT

A boilermaker's assistant aged 58 years was admitted in a vague confused state after collapsing at work. He denied any previous faintness, giddiness or undue fatigue. He said he was not short of breath on exertion, he had no dyspepsia and ate well. The bowels were normal and he had not noticed any change in the appearance of the motions. His mental acuity was subnormal throughout the period of observation. According to his workmates this was his usual mental condition.



FIG. II. Median vertical section of the polyp. Vascular submucosal tissue forms its core, at some points penetrating the overlying mucosa.

Striking pallor was the outstanding physical feature. His tongue was very pale, atrophic and fissured. Abdominal examination was normal and no skin haemangiomas were present.

Blood examination revealed a microcytic, hypochromic anaemia with a haemoglobin of 6.6 gms. per 100 ml., a red cell count of 2.3 million per c.mm. and 3 per cent. reticulocytes. There was a mild neutrophil leucocytosis with a total white cell count of 13,000 per c.mm. A well marked normoblastic hyperplasia was present in the sternal marrow.

Tests for occult blood in faeces were persistently positive so investigations were directed towards determining the cause of the bleeding. Barium meal examination was normal. Barium enema examination revealed numerous diverticula in the colon. However, in the absence of any bright bleeding, this

was regarded as unlikely to be the source of the bleeding. Gastroscopy and gastric biopsy were normal.

Blood transfusion failed to maintain the haemoglobin level and tests for faecal occult blood continued positive. Repeated blood transfusion presented a special problem in this patient since his blood group was B3 Rh negative. Laparotomy was therefore decided upon. At operation blood was seen in the small intestine. The stomach and duodenum were externally normal and on opening into them no lesion could be found. The full length of small bowel was examined three times, and only at the third attempt at a point 18 inches from the duodeno-jejunal flexure a small, soft thickening was felt. On opening the bowel, this proved to be a small bleeding polyp. A local resection was performed.

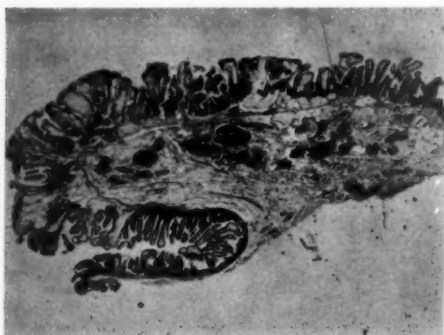


FIG. III. Photomicrograph showing dilated vascular channels which occupy the submucosa and some areas of the mucosa. Some channels contain blood and others eosinophilic coagulum. Stain haematoxylin and eosin. (X 8).

The polyp measured 10 mm. in length and 5 mm. in diameter. On sectioning longitudinally it had a highly vascular core occupying the submucosal layer of the bowel wall. There were a number of points where haemorrhage had burst through the overlying mucosa on to the surface (Figs. I and II). Microscopically (Fig. III) the tumour was situated mainly in the submucosal layer but also extended into the mucosa. It was made up mainly of cavernous endothelial-lined spaces, many packed with red cells but others containing only an eosinophilic coagulum. Among the cavernous spaces were small arteries and, particularly at the base, some irregular blood channels with walls of mixed smooth muscle and fibrous tissue. No collections of lymphoid tissue were present. The lesion was considered to represent a mixed lymphangioma-haemangioma.

The patient's post-operative course was initially satisfactory except that his dull mental state produced nursing difficulties. A sacral bed sore appeared but was healing satisfactorily when he developed pneumonia, and a few days later he died.

Autopsy confirmed the presence of pneumonia and also revealed a further jejunal polyp of about the same size as the one removed at operation, but pale, not haemorrhagic in appearance. Microscopically, it consisted of dilated cystic spaces lined with endo-

thelium and containing an eosinophilic coagulum without any red cells. Occasional small collections of foamy macrophages were present within the spaces. Numerous arteries, veins and irregular blood channels were intermingled with the lymphatic spaces. The tumour was regarded as essentially similar in type to that removed at operation.

COMMENT

Angiomata of the intestine are rare tumours. Both haemangioma and lymphangioma occur. Heycock and Dickinson (1951), in a review of intestinal haemangioma, were able to collect 85 cases since the first report by Gascoven in 1860. In 44 cases the lesions were single and in 41 they were multiple. Lymphangioma are of far less frequent occurrence. They are briefly mentioned in an extensive review of tumours of the small intestine by Raiford (1932), and Naumann (1927) reports one case and gives references to others. The occurrence of mixed haemangioma and lymphangioma is noted by Landing and Farber (1956), and by Willis (1948). These authors state that the mixed type of lesion is particularly common in the region of the upper part of the neck, the pharynx and the parotid gland. No mention, however, is made of their occurrence in the intestine, nor are mixed lesions described in the cases reported by Heycock and Dickinson (1951) and by Naumann (1927).

In the present case it was the blood-vascular component that constituted the clinically significant element — bleeding from this leading to his presentation with anaemia. Intestinal haemangioma most frequently present in this way. The anaemia is characteristically insidious in onset and in several of the reported cases had been present a considerable time before the diagnosis was made. When haemangioma are multiple, more rapid blood loss may occur, and such cases constitute a great surgical problem. However, in the case reported by Heycock and Dickinson (1951) a total of 30 separate haemangioma were successfully removed at two separate operations.

Another mode of presentation of haemangioma is as an intestinal obstruction brought about either by narrowing of the lumen as occurs where there is a diffuse infiltration involving all layers of the bowel wall (Brown, 1924), or by producing an intussusception as may occur with a polypoid growth. Two instances of the latter are reported by Merchant (1939). One in a boy aged 8 was associated

with multiple haemangiomas of the skin and viscera; the other was found in a woman aged 67 who died from peritonitis due to a ruptured gall-bladder.

Rarely, symptoms simulating peptic ulceration have been the presenting feature of a haemangioma as in the case reported by Dudley (1934) where the patient had multiple sub-serosal haemangiomas throughout the length of the small intestine.

Symptoms initially diagnosed as salpingitis were the presenting feature in the 23-year-old woman with a lymphangioma of the ileum described by Naumann (1927). A tumour 1.5 cm. in diameter was located in the mucosa and submucosa. Microscopically it consisted of cavernous endothelial-lined spaces containing fluid but no blood. A larger extension into the mesentery was also present. This was a loculated cyst containing 40 ml. of chyle.

Lymphangiomas of appropriate growth pattern could presumably cause intestinal obstruction as sometimes happens with haemangiomas. However, undoubtedly the most likely serious complication of an angioma is haemorrhage. Sometimes intestinal haemangiomas are associated with similar tumours at other sites. The original patient described by Gascoyen (1860) had lesions in the bowel, liver, parotid and skin. The presence of these skin tumours in an obscure case of alimentary bleeding is a strong pointer to a haemangioma. However, it is a diagnosis to be considered in any such case where no clinical or radiological abnormality is demonstrable and warrants careful search at laparotomy for what may prove to be an inconspicuous yet critical lesion. In addition, search for further tumours is indicated when one is found, since multiple lesions have been present in 48 per cent. of reported cases of haemangiomas.

SUMMARY

A case report is presented of a 58-year-old man with severe anaemia due to bleeding from a mixed lymphangioma-haemangioma of the jejunum.

The mode of clinical presentation of small intestinal angiomas is discussed.

In obscure cases of alimentary tract bleeding rare causes need to be kept in mind and a careful search made for small lesions at laparotomy.

ACKNOWLEDGEMENTS

I wish to thank Mr. Julian Orm Smith, who performed the operation, and Dr. Ian Wood for permission to publish this case report, and for their helpful comments on its presentation. I am indebted to Dr. J. D. Hicks for discussion on the pathology of the tumour and for the photomicrograph.

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THE MECHANISM AND NATURE OF THE INJURY IN DISLOCATIONS OF THE ELBOW AND A METHOD OF TREATMENT*

By P. I. CROMACK

Perth

DISLOCATION of the elbow is a common traumatic incident and is more often seen in children and adolescents, where it is the most likely joint to dislocate, due to both the coronoid and olecranon processes being imperfectly formed (Key and Conwell, 1942).

The injury, both in children and adults, is sometimes followed by persistent disability and is chiefly shown by a loss of extension to a lesser or greater degree and also a reduction in the range of rotation of the forearm. This disability is observed not only in those joints where the X-ray shows significant bony damage, such as a fracture of the medial epicondyle or radial head but also, in those cases where the bony damage is revealed merely by an avulsed flake of bone, usually from the epicondylar region.

the common flexor origin. It is my contention that this "fibrosis" is responsible for the persistent loss of movement which may occur and that it can be prevented by surgical repair in carefully chosen cases.

Incidence

From Table 1 it can be seen that the occurrence of significant disability is high, 65 per cent., following this injury. The commonest type of dislocation is the posterolateral dislocation of the joint as shown in Fig. 1.



FIG. 1. Radiographs showing the common posterolateral dislocation of the elbow.

TABLE 1
DISLOCATION OF THE ELBOW

Incidence of types of dislocation

(Children and adults: 97 cases).

Number of cases = 33 chosen for review

(No significant bony damage in any of these).

Analysis of types in group

67 per cent. posterolateral dislocations	= P.L.
27 per cent. posteromedial	= P.M.
6 per cent. pure posterior	= P.

Number treated conservatively

Total = 23.

Out of this 15 or 65 per cent. had significant disability on discharge.

If, following a dislocation, the joint be examined between the sixth and eighth week, it is often possible to feel considerable thickening of the soft tissues about the joint, particularly on the inner side in the region of

My interest in this injury has been stimulated by the absence of exact information on the subject in the literature and, more recently, by the naked eye inspection of the site of trauma itself. Attention has been focussed chiefly upon the posterolateral dislocation since not only is this the commonest type, but because the indications for surgery are more pressing. It is this type of injury which may produce the greatest disability. Owing to the age incidence, most of the cases in this series are children.

*Received for publication 22nd March, 1960.

NATURE OF THE INJURY

It is stated that dislocation of the elbow is necessarily associated with avulsion of the brachialis from the coronoid process (Watson Jones 1956). This is not found to be so in posterolateral dislocations of the elbow.

The soft tissue tear begins at the common flexor origin, which is usually displaced with a fragment of bone, consisting of the whole or part of the medial epicondyle. Thus begun, the rent continues, tearing through the periosteum and stripping off the humeral attachment of pronator teres. This muscular attachment usually remains with the epicondylar segment and these two structures, together with the humeral attachment of the medial collateral ligament, are displaced together downwards and medially.

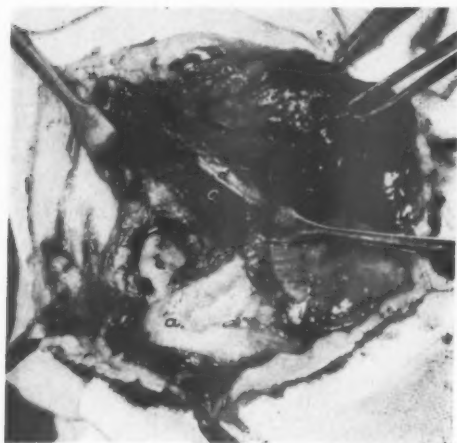


FIG. II. Medial approach. Skin, fat and fascia only divided. No other dissection performed.

- (a) Common flexor origin displaced downwards.
- (b) Bared bone of the pronator teres origin.
- (c) Extensive tear in muscle fibres of brachialis held open by retractors — distal portion of muscle still attached to coronoid.
- (d) Median nerve lying free in wound.

At the site of the detachment of pronator teres, the humerus is bared for a small area, perhaps $\frac{1}{2}$ in. x $\frac{1}{4}$ in., since the proximal edge of the periosteum retracts and tends to roll upon itself leaving the bone bare.

The soft tissue tear has now reached the intermuscular space between the pronator teres and the fibres of the brachialis coursing down to their insertion. The rent spreads

laterally through the anterior capsular attachment until the joint cavity is laid open in continuity with the soft tissue tear.

If the forces continue to act, the medial fibres of the brachialis are stretched across the sharp medial condylar region and torn through, so that as much as 50 per cent. of the muscle may be divided (Figs. II and III). This muscle is not avulsed from bone.



FIG. III. Soft tissue rent showing.

- (a) Avulsed attachment of common flexor origin.
- (b) Raw bone area and the sharp edge as described in text lying superiorly.

Thus a large rent is left in the medial side of the joint which is roofed over merely by skin fat and fascia. When these structures have been divided, the median nerve, which shields from view the brachial artery, can be seen crossing the lateral extremity of the soft tissue defect.

MECHANISM

Most writers (Watson Jones, Wilson, Key and Conwell) describe a fall upon the outstretched arm, with the elbow fully extended, as the mechanism producing dislocation of this joint. It would appear that upon the basis of these findings and examination of the elbow under anaesthesia, that this is not the common mechanism.

On falling to the ground, the common protective mechanism is to flex the elbow to

about 145° and pronate the wrist. The arm and elbow in this position are in the best position to receive the impact of the body weight and act as a shock absorber. If the force proves too great for the mechanism a posterolateral dislocation of the elbow results.

In this position of pronation and flexion, the common flexor muscles are taut, and it is upon them that the force first acts. As the palm of the hand meets the ground and the rest of the body slides away, the humerus is internally rotated in relation to the fixed forearm, while the elbow joint is also subjected to an abduction strain.

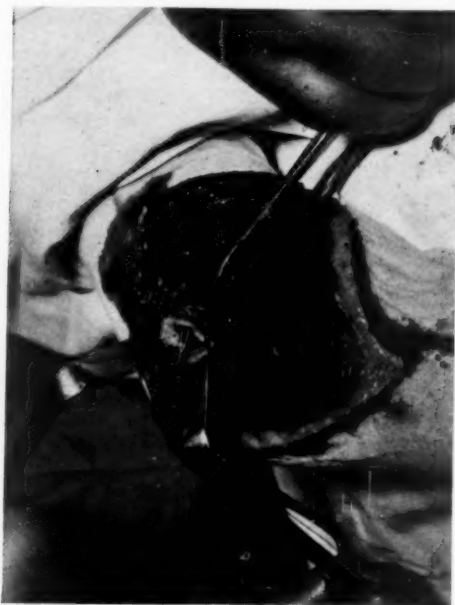


FIG. IV. Placement of drill holes in re-attachment of flexor origin.

With the avulsion of the common flexor origin, the tear continues through the medial ligament of the joint and the anterior capsule. The joint is now unstable (as can be proved during anaesthesia) and the humerus may now easily slide forwards out of the olecranon fossa, producing the posterior displacement, while the head of the radius displaces laterally or posterolaterally. This is the common posterolateral dislocation of the joint.

The less common simple posterior dislocation arises by a fall upon the outstretched arm with the elbow in full extension forcing

the humerus out of the olecranon. It is because the humerus simply slides out of the olecranon socket that the soft tissue damage appears to be of a lesser degree. This type of injury appears more commonly in adults.

Posteromedial dislocations are rare, are probably due to direct violence, and will not be discussed here.



FIG. V. Flexor origin re-attached. Capsular rent being repaired. Brachialis tear not yet repaired.

ASSESSMENT

With full realization of the extent and variation of the soft tissue injury, it becomes clear that conservative measures may not succeed in every case in restoring full function. First it is necessary to distinguish those injuries which require surgical repair. There are three special features to consider. (i) An X-ray showing the nature of the displacement serves only to remind one that careful clinical examination is necessary. (ii) If, in a posterolateral dislocation with a significant tear, the medial and anterior epicondylar region be palpated early before the haematoma becomes firm, the impression will be gained that the bone in the region is merely covered by skin. This is so. (iii) The diagnostic procedure is an examination under anaesthesia. When the elbow is reduced, as it must be, the

degree of soft tissue tear is easily assessed, if this has not already been done, as it may be, with experience, before reduction. If the soft tissue tear is significant, with the elbow flexed to 145° and the hand pronated, external rotation and abduction of the forearm reproduces the dislocation very easily. This is the type of injury requiring surgery.



FIG. VI.

It is interesting to find that the medial epicondyle is rarely trapped in the joint if the soft tissue tear is extensive.

REPAIR

The surgical repair, if decided upon, should be performed in the first few hours, when it is still possible to distinguish each structure separately and when they are more easily amenable to surgical repair. There were no cases of myositis ossificans in this series and this may be due to an immediate repair before the haematoma commences to undergo biochemical changes.

The supine position of the patient, with the elbow flexed and lying on a hand table, will be found the easiest position in which to operate. An incision in the long axis of the limb along the medial side of the joint, dividing the skin and fascia only, will be found to lead directly to the rent, which is immediately obvious in the correctly chosen case. There is no need for further dissection other than to verify the position of the ulnar nerve.

It is easiest to conduct the repair in the following order:

(i) Epicondyle and attached muscles

Unless the bony fragment carries with it any articular cartilage, which is unusual, it is best to shell out the bony kernel which leaves the flexor muscles attached to a tough morsel. This fragment is sutured direct to the raw bone by means of two very small drill holes. By so doing, a painful swollen epicondylar protuberance, persisting for many months, is avoided and perhaps late traumatic ulnar neuritis. The humeral attachment of the medial collateral ligament is usually found attached to the morsel (Fig. IV).

TABLE 2
RESULTS OF SURGERY

Cases	Type	Age	Time to Function (i.e. full flexion to full extension, with full rotation)
J.H.	P.L.	13 yrs.	11 weeks
P.H.	P.L.	12 "	8 "
A.W.	P.L.	28 "	10 "
A.F.	P.L.	11 "	10 "
J.P.	P.L.	14 "	14 "
P.C.	P.L.	15 "	12 "
P.H.	P.L.	10 "	10 "
A.H.	P.L.	12 "	8 "
H.C.	P.L.	11 "	12 "
E.W.	P.L.	11 "	10 "
I.H.	P.L.	9 "	10 "
P.W.	P.L.	12 "	8 "
D.W.	P.L.	13 "	9 "
L.S.	P.L.	10 "	13 "
B.J.	P.L.	11 "	8 "

(ii) The anterior capsular attachment is repaired with a purse-string suture (Fig. V).

(iii) The brachialis tear, if present, is then repaired by fine sutures.

(iv) The periosteal cuff is replaced.

It only remains to repair the fat and fascia and suture the skin. The elbow should be

immobilized in plaster at 90° for 3 or 4 weeks and then allowed gently to move. The recovery period to full function is 6 or 8 weeks

following the removal of the plaster. Figs. VI to IX show the clinical results obtained (cases P.H. and J.P. from Table 2).



FIG. VII.

Range of movement of case P.H. 8 weeks after operation.



FIG. VIII.

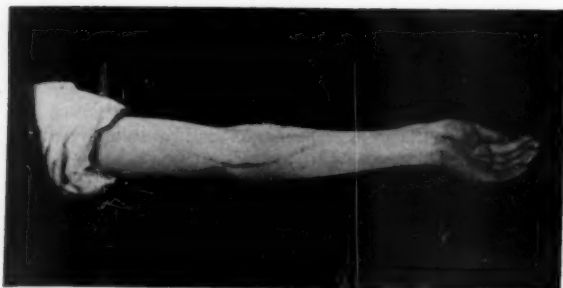


FIG. IX.

Similarly clinical results of case J.P. 14 weeks after operation.

SUMMARY

The importance of the soft tissue injury in some types of dislocation of the elbow is outlined. Healing by scar tissue may well be responsible for the poor results which occur in an injury which appears to be simple. By an exact surgical repair in selected cases, the anatomy can be reconstituted allowing rapid healing and recovery of full function.

ACKNOWLEDGEMENTS

I am grateful for the help and encouragement given to me by Mr. F. G. Badger at the Birmingham Accident Hospital at the commencement of my interest in this subject. I am also indebted to the Department of Medical Photography, Royal Perth Hospital, for their technical assistance.

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ACUTE EMPHYSEMATOUS CHOLECYSTITIS*

REPORT OF A CASE

By G. W. TRINCA

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ACUTE emphysematous cholecystitis is an uncommon form of cholecystitis characterized by the accumulation of gas in the gall-bladder and subsequent extension of the gas into and beyond the wall of that organ. Infection by gas-producing organisms, of which the clostridia form the largest group, is responsible for this gas formation.

Edinburgh and Geffen (1958) have reviewed 50 cases reported up to 1958 since the condition was first described at autopsy in 1901. They report in their review that the condition was first diagnosed at operation in 1908 and that it was not until 1931 that a pre-operative diagnosis was made on radiological grounds. Wilson (1958) has reviewed 26 cases reported between 1908 and 1951 and has described a case he treated without surgery. He makes the point that the condition is not described in any detail in most textbooks of surgery.

Hamilton Bailey (1959) does describe the condition of acute emphysematous cholecystitis. The signs and symptoms are those of severe acute cholecystitis, the patient often being a diabetic. He advises cholecystectomy as the treatment of choice. Byrne (1959) mentions clostridia as a source of infection giving rise to gas in the gall-bladder and states that the infection is often associated with diabetes.

Generally, however, surgical textbooks do not separate emphysematous cholecystitis as a clinical entity, although mention is made of clostridial infection being one of the rarer forms of infection in cholecystitis. Bailey and Love (1956) state that if the infection is due to *Clostridium welchii* it is usually of a fulminating nature. On the other hand, Whipple (1935) points out that *Clostridium welchii* can exist in the gall-bladder and biliary tree without causing symptoms. Christopher (1946) states that clostridium *welchii* can be

isolated from the wall of the gall-bladder and the bile in apparently normal gall-bladders. Gordon-Taylor and Whitby (1932) investigated Clostridial infection in the gall-bladder and found that the organisms are more often found in calculi and gall-bladder wall than in the bile. These organisms were found to be of low virulence and non-sporulating. They contended that the liver and bile radicles were a normal habitat for gram positive anaerobic organisms and a media was established if there was injury or anoxia of the gall-bladder wall.

It is generally accepted that the majority of cases of acute cholecystitis are associated with calculi (85-95 per cent.), the initial process being obstruction of the cystic duct or neck of the gall-bladder. Infection, when it occurs, is a sequela of the pathological changes occurring as a result of the obstruction. This is held for all organisms, including clostridia, which can exist for a long time in the bile or wall of the gall-bladder without producing symptoms. The commonest organisms encountered are the streptococci and *B. coli*. Inspissated bile can cause obstruction of the cystic duct in the absence of calculi.

Acute cholecystitis without obstruction is uncommon, and is considered to be due to bacteria which have reached the gall-bladder by one of a number of routes, the blood stream being the most favoured. Shackelford (1955) considers that the inflammatory swelling of the mucosa of the gall-bladder and cystic duct that occurs in these cases of non-calculus cholecystitis eventually results in obstruction of the duct and that this is followed by the same sequence of events that occurs in acute obstructive cholecystitis.

Edinburgh and Geffen, whose review is more comprehensive than Wilson's, make the following observations. The average age of the patients was 59 years and males predominated (72 per cent.). There was frequently

*Received for publication 12th December, 1959.

a previous history of biliary colic and gall-bladder disease. Diabetes was detected in 24 per cent. and jaundice occurred in 20 per cent. of cases. Calculi were found in 80 per cent. of cases investigated for calculi. Clostridia were isolated in 40 per cent. and *B. coli* in 30 per cent. of cases. In 44 per cent. of cases a mass was palpable. Wilson makes similar observations, although he states that in those cases where organisms were isolated it was usually one of the clostridial group.

Although no cases have been reported in this country, it seems certain that cases of this type of cholecystitis have occurred and have either not been reported, or there has been no pre-operative diagnosis made on radiological grounds.

In the case to be reported there are a number of features which are neither typical of acute cholecystitis generally nor fit into the clinical picture of acute emphysematous cholecystitis.

CASE REPORT

N.C., male, aged 37 years, was admitted to Prince Henry's Hospital on 26 May, 1959, with the provisional diagnosis of acute emphysematous cholecystitis, pleural effusion and possible sub-phrenic abscess.

The history obtained on admission was as follows. He had been perfectly well until six days previously when he had developed central abdominal pain and vomiting. The pain was also referred to the lower abdomen and was neither colicky nor very severe. The pain subsided in a few hours but his nausea and anorexia persisted. Two days later the pain recurred in his right upper abdomen and this was aggravated by deep breathing and coughing. There was also some shortness of breath. His local doctor was called in at this stage. Dr. Frank Misell who attended the patient for the four days prior to his admission sent the following notes when referring him to hospital.

Temperature was 99 F. Examination of his chest revealed diminished breath sounds in the right lower chest. There was slight tenderness in the right hypochondrium but no rigidity and there was no mass to be felt. He was considered to be suffering from a right basal lung infection and was prescribed penicillin and achromycin. He was seen again two days later when the pain and dyspnoea had lessened, although his cough persisted and a small amount of clear sputum was produced.

On the day prior to admission his temperature was 102 F. There had been no vomiting for three days. Examination revealed dullness and absent breath sounds at the right base. Tenderness persisted in his right hypochondrium but this was not marked.

Arrangements were made for X-ray of his chest and a plain film of his abdomen and following the radiological report he was admitted to hospital.

Further relevant facts obtained from the patient after admission were as follows.

His digestion had always been good and he had not experienced previous abdominal pain. There was no intolerance to fatty foods. His weight was steady and he neither smoked nor drank alcohol. Bowels were always regular and there had been no disturbance of micturition, although his urine had been a little darker for three days prior to admission. He had never had any previous chest trouble.



FIG. 1. Plain film of right upper quadrant taken prior to admission. Note gas-filled gall-bladder with infiltration of wall and extension of gas beyond.

On admission examination revealed a healthy looking, well-nourished man in no apparent distress. Temperature 101.4 F. Pulse 88, regular and good volume. Respiratory rate 24. Blood pressure 150/90 mm. of mercury. Vessels not palpable. Urine S.G. 1018, acid reaction, trace of albumin, no sugar, diacetic acid or acetone.

Cardio-vascular and central nervous systems. No abnormality detected.

Chest: Good movement, abdomino-thoracic respiration. Signs of pleural effusion at right base.

Abdomen: Lax and moved with respiration. McBurney scar present. Moderate tenderness in the right hypochondrium. Murphy's sign was positive. No mass palpable.

No abnormality detected on rectal examination.

The radiological report (Fig. 1) which accompanied the patient was as follows:—"Gall-bladder region. The gall-bladder is distended with gas and the gas has infiltrated the gall-bladder wall. This

constitutes the condition of acute emphysematous cholecystitis. The common bile duct is not gas-filled but there are a few blebs of gas above the gall-bladder suggesting that infection has spread to the surrounding tissues. The liver has not been displaced.

Chest: The lower part of the right lung field is obscured by a homogeneous density which represents pleural fluid. This makes it impossible to determine whether the right hemi-diaphragm is elevated or not. The remaining lung fields are clear."



FIG. II. Film taken after aspiration of pleural cavity but before operation. There has been extension of gas well beyond the gall-bladder. Note subphrenic collection of gas and the fluid level in the gall-bladder. There is elevation of the right hemi-diaphragm and right lower lobe collapse.

Further management. A diagnosis of acute emphysematous cholecystitis with pleural effusion and probable subphrenic abscess was made.

It was decided to carry out a number of procedures and investigations before embarking on surgical relief of his condition. Accordingly, arrangements were made for aspiration of his effusion to be followed by screening in order to determine the position and movement of his right hemi-diaphragm.

Two hundred cc. of thin brown non-odorous fluid were removed from his right pleural cavity. This fluid did not clot. A smear of the pleural fluid

showed amorphous debris, degenerate polymorphs but no bacteria. The fluid was also cultured but subsequently no growth was obtained.

Screening of chest was performed following pleural aspiration and the right hemi-diaphragm was seen to be raised and immobile. This strongly suggested a subphrenic abscess.

Additional investigations carried out at this time were:

White cell count, 20,000; urine, no bile salts or pigments, urobilinogen — 0.48 mgm. per cent.; casoni, immediate and delayed reactions were negative.

Treatment at this stage consisted of oral fluids, penicillin 500,000 units intramuscularly 6 hly., streptomycin 0.5 gm. b.d. and pethidine 50 mgm. 6 hly. if necessary.



FIG. III. Enlargement of Fig. II showing gall-bladder in greater detail.

On the morning after admission (27 May) the condition of the patient was good. His temperature was normal and his pulse was 80 per min. He still complained of some pain in his right upper abdomen and there was some radiation of this pain to his right lower quadrant. An X-ray of his chest and plain film of his abdomen (Figs. II and III) were carried out. The report was as follows:—"Abdomen. Films in different positions confirm the presence of a gas and fluid-filled gall-bladder having gas infiltrating its walls consistent with the presence of infection due to a gas-forming organism. These films further show the presence of subphrenic gas bubbles indicating the spread of infection to those regions and thence presumably to the pleura."

Results of liver function tests carried out pre-operatively were as follows:—

Total serum protein 6.9, albumin 3.5, globulin 3.4, A/G ratio, 1.0; serum bilirubin 0.7 mgm. per cent.; alkaline phosphatase 27 units; gamma globulin 2 units; cephalin flocculation 24 hours +, 48 hours ++.

Despite the obvious improvement in the patient's condition operation was performed on the afternoon of 27 May, i.e., eighteen hours after admission. The presence of a gas infection involving the gall-bladder with extension beyond that organ giving rise to a subphrenic abscess and right pleural effusion indicated surgical intervention to drain the subphrenic and pericholecystic abscesses and if possible remove the diseased gall-bladder.

Details of operation: Operation was performed under general anaesthesia (Pentothal induction, relaxant with intubation, nitrous oxide and oxygen).

The abdomen was opened through a right Kocher's incision and operative findings were as follows:—

1. Numerous friable adhesions between diaphragm and superior and posterior surfaces of the liver.

2. Copious thick yellowish-green pus in the right suprahepatic and infrahepatic subphrenic spaces.

3. A large leaking pericholecystic abscess walled off by omentum, liver and adherent oedematous duodenum. Thick pus and bubbles of gas observed escaping from this mass.

4. Colon not attached to the mass and no abnormality detected in the stomach.

Operative procedures carried out were as follows: Adhesions between liver and diaphragm broken down and pus aspirated from the subphrenic spaces. Swab of pus taken for culture. Omentum and duodenum freed from gall-bladder mass by blunt dissection. The gall-bladder wall was swollen, friable, necrotic and fragmenting in places. Crepitus and gas bubbles were present. The outer wall of the gall-bladder was separated completely from the mucosa which was distended and appeared intact. The mucosa was dark grey in colour.

Retrograde cholecystectomy was attempted and the intact mucosa was dissected by blunt and sharp dissection until the cystic artery was identified and ligated. Removal had proceeded as far as the neck of the gall-bladder when the mucosal wall disintegrated in a necrotic mass of friable material. The neck of the gall-bladder was clamped and ligated. It was technically impossible to identify the cystic and common bile ducts.

During removal of the gall-bladder the cystic artery ligature slipped. Haemostasis was secured by picking up the vessel with plain dissecting forceps and using diathermy.

The gall-bladder contained a thick green sludge but no calculi were seen.

The gall-bladder bed was drained with a soft rubber tube emerging from the lateral end of the abdominal incision. The subphrenic space was drained with a soft rubber tube in the right hepatic space and emerging through a separate stab wound in the loin.

The peritoneum was closed with continuous interlocking catgut and the right rectus sheath and skin approximated with interrupted chromic catgut and linen respectively.

Post-operative management: The patient was given nothing orally and intravenous fluids were administered consisting of two pints of whole blood, each in six hours, followed by one litre of normal saline and two litres of 5 per cent. dextrose in water over the following twenty-four hours. Penicillin and streptomycin therapy was continued.

On the day following operation, the patient felt and looked well. Temperature was normal and haemoglobin estimation was 105 per cent. Bowel sounds were present and he tolerated oral fluids well. Intravenous fluid administration was discontinued.

Subsequent progress was satisfactory apart from a slight elevation of temperature for a few days. There was a moderate amount of bile-stained fluid from the tube draining the gall-bladder bed and a slight serous ooze from the other drain tube. The tubes were removed on the twelfth post-operative day.

Streptomycin was discontinued on 8 June, and penicillin on 15 June.

The patient sat out of bed on the fourth post-operative day and was discharged from hospital on 20 June, twenty-four days after admission.

Results of investigations carried out in the post-operative period were as follows:—

X-ray chest (29 May): "Chest structures grossly deformed by kypho-scoliotic deformity but the right hemi-diaphragm is still a little elevated and fixed by lateral costo-phrenic adhesions. The left lung is clear apart from plate atelectasis at the left base. Findings are collapsed right lower lobe and resolving subphrenic lesion."

Swab of subphrenic pus: *Clostridium welchii* identified. Very sensitive to penicillin, chloromycetin and tetracycline. Sensitive to erythromycin; resistant to streptomycin.

Biopsy report of gall-bladder: (a) Macroscopic. Free irregular pieces of tissue all of which are variegated in colour. Colour varies from yellow to greenish red. Only a portion of it is recognizable as gall-bladder and it is all very friable. (b) Microscopic. Section shows gross inflammation with necrosis and destruction of the gall-bladder wall. Conclusion. Severe acute cholecystitis."

X-ray chest 1 June: Right lower lobe collapse. (The patient was clear of chest signs on 11 June.)

Barium meal 18 June: "Apart from a small diverticulum approximately 1 cm. in diameter projecting from the third part of the duodenum, no abnormality of the upper alimentary tract is detected."

Intravenous cholelithogram 20 June: "One film shows a small aggregation of dye in the estimated site of Hartmann's pouch and the upper portion of a moderately dilated common bile duct (12 mm. in diam.), so that there is no radiographic evidence in these films of the presence of a biliary fistula."

The patient was seen in the Outpatient Department on 26 June and 10 July, when he stated that he felt well and was free from chest and abdominal symptoms. He resumed work on 24 July, 1959, approximately eight weeks after the onset of his illness. He was seen finally two weeks after he resumed work and at that time was in good health and apparently fully recovered.

DISCUSSION

A case of acute emphysematous cholecystitis has been presented.

The onset and course were not typical of acute obstructive cholecystitis, presenting more as a right basal lung infection. Prior to admission to hospital the patient was treated for a respiratory infection. The development of a right pleural effusion prompted radiological investigation of the chest. As the attending medical practitioner suspected an associated intra-abdominal condition a plain X-ray film of the abdomen was also carried out. Pre-operative diagnosis was thus made on radiological grounds.

The importance of excluding non-surgical conditions in the diagnosis of the acute abdomen has been stressed by Cope (1946). As shown in this case, the reverse equally applies. The accurate diagnosis of acute cholecystitis is not always easy, as other intra-abdominal conditions can simulate this disease, e.g., peptic ulcer, pancreatitis, right pyelonephritis (Byrne, 1959). A plain film of the abdomen is therefore of importance in arriving at a correct diagnosis.

In this case, if an X-ray had not been carried out, the patient may have been treated for a pleural effusion assumed to be due to underlying lung pathology and a dangerous delay would have occurred in the treatment of the intra-abdominal condition.

The absence of calculi in this case is unusual. There was no past history of biliary colic or symptoms suggestive of gall-bladder disease. It is possible that infection occurred with other organisms resulting in damage to the gall-bladder wall and this then provided the necessary media for the multiplication of *Clostridium welchii* which had been existing for some time in an otherwise normal gall-bladder. Alternatively, inspissated bile may have been the obstructing factor that set the process going.

Although diabetes was not present in this case, the relationship of diabetes and emphysematous cholecystitis is sufficiently strong to warrant investigation for diabetes in cases of acute cholecystitis.

The value of a plain film of the abdomen in the diagnosis of acute cholecystitis is well illustrated in this case. The presence of calculi or gas in the gall-bladder in the presence of the clinical signs and symptoms of acute cholecystitis is valuable confirmatory evidence and may be of considerable help in those cases where diagnosis is difficult. In this case the type and degree of infection was known before operation. A pre-operative diagnosis of emphysematous cholecystitis would enable early and adequate administration of antibiotics. The lack of toxicity in this case may well be due to the fact that he received antibiotics for some days prior to operation. It was subsequently found that the *Clostridium welchii* isolated was sensitive to these drugs.

In Edinburgh and Geffen's review it was seen that virulent infection occurred in those cases treated in the pre-antibiotic era or who did not receive antibiotics when those drugs were available.

Altemeier and Furste (1949) investigated the virulence of *Clostridium welchii* in guinea pigs. Great difficulty was encountered in provoking a gas infection. When infection did occur it was in the presence of dirt and damaged muscle. In the absence of damaged muscle tissue, clostridial infection in the gall-bladder need not run a fulminating course. Virulence is no more a predominant property of *Clostridium welchii* than it is with any other organism. However, once operation has occurred in a case of acute emphysematous cholecystitis due to clostridial infection, damaged muscle is present and a favourable medium has been established for a serious gas infection in muscle. The pre-operative diagnosis of emphysematous cholecystitis is therefore most important. It is equally important that the condition be recognized at operation in those cases that have not had pre-operative radiological investigation so that effective antibiotic therapy can be instituted. The presence of a pericholecystic abscess with separation of the outer gall-bladder wall from a gangrenous mucosa in the presence of crepitus and obvious gas bubbles indicates infection of the gall-bladder by gas-forming organisms.

The value of anti-gas gangrene serum is doubtful. It was not given in this case as the disease had been present for some days and the patient was not toxic.

The radiological diagnosis of emphysematous cholecystitis is straight-forward. In the early stages gas is seen only in the gall-bladder but this is followed by a darker translucent ring surrounding the gall-bladder and due to gas contained in the wall. In the erect position there is usually a fluid level (Figs. II and III) present. There is no gas in the bile ducts. Radio-opaque calculi may be seen. Later the gas extends beyond the gall-bladder. In this case, the films show gas passing through the gall-bladder wall and also under the diaphragm (Figs. II and III).

Other conditions in which gas can occur in the gall-bladder are an internal biliary fistula, operative or pathological, and in cases of incompetence of the sphincter of Oddi. With a fistula, gas is also seen in the bile ducts and there is no gas in the gall-bladder wall and no fluid level can be demonstrated. Barium meal or enema may demonstrate a communication between the gall-bladder and stomach or intestine.

With an incompetent sphincter, gas would be demonstrated in the ducts.

Lipomatosis of the gall-bladder may simulate gas in the gall-bladder. However, this condition does not present as acute cholecystitis. Radiologically there is no change in the configuration of the translucent shadow with change in position and a fluid level cannot be demonstrated.

The problem of when to operate applies to acute emphysematous cholecystitis as much as it does to the more usual form of acute obstructive cholecystitis. The condition does appear to run a less severe course and this would favour conservative treatment in the acute phase. Both Edinburgh and Geffen and Wilson in their reviews suggest that the patients do not appear to be desperately ill and that there is a place for conservative treatment, particularly if the patient is first seen three to four days after onset of the disease.

The frequency of a pericholecystic abscess with gross tissue destruction is much greater in this type of cholecystitis, the mass becoming evident about the third to fifth day (44

per cent.). Extension of the infection, as occurred in this case, seems more likely than in the usual form of obstructive cholecystitis.

An increasing mass in an otherwise improving patient in whom emphysematous cholecystitis has been diagnosed radiologically is not necessarily an indication for operation. In this case, the indication for operation was the considerable extension of infection beyond the gall-bladder giving rise to a large sub-phrenic abscess and pleural effusion.

It would seem, therefore, that if this condition is diagnosed within the first forty-eight to seventy-two hours, operation with cholecystectomy and antibiotic therapy is the treatment of choice. If the condition has been present for more than three to four days and the patient's condition is good, conservative treatment with antibiotics is justified, even in the presence of a mass. As with conservative treatment of all forms of cholecystitis the surgeon must be prepared to operate if there is any deterioration in the patient's condition. The importance of a plain X-ray film of the right upper quadrant as a diagnostic measure and as an aid to the progress of a conservatively treated case, cannot be too strongly stressed.

In those cases treated conservatively in the acute stage, it is suggested that elective cholecystectomy should be performed at a later date, viz., ten to twelve weeks. It is interesting to note that in the case treated by Edinburgh and Geffen a large inflammatory mass was present in the right sub-hepatic space at operation six weeks later. A thick-walled abscess containing a little pus was drained. Culture produced *B. coli*, *B. lactis aerogenes*, *B. pyocyaneus* but no clostridia. There was bile drainage for twelve days post-operatively. Jemerin (1949) in reporting his case, delayed operation two weeks. Penicillin was given pre-operatively but at operation a pericholecystic abscess was found from which clostridium oedematiens was isolated.

This would suggest that in acute emphysematous cholecystitis a pericholecystic abscess is a common occurrence and that in those cases treated conservatively resolution of that abscess is slow and may never be complete. This could lead to a state of chronic ill-health in a person so afflicted.

The ideal management in a case of acute emphysematous cholecystitis is to diagnose the condition as early as possible so that cholecystectomy can be carried out before the development of a pericholecystic abscess and subsequent extension of infection beyond the confines of that abscess. In those cases treated conservatively the same criteria for surgical intervention hold as for all types of acute cholecystitis and in those cases that have responded to conservative treatment it is imperative that definite plans be made for elective cholecystectomy at a later date.

SUMMARY

1. A case of acute emphysematous cholecystitis due to *Clostridium welchii* has been reported. Unusual features in this case have been commented upon and it is suggested that this condition occurs more frequently than is indicated by the medical literature.

2. Pre-operative plain X-ray of the abdomen with special reference to the right upper quadrant is a valuable investigation in the diagnosis of acute cholecystitis and is diagnostic in that form of cholecystitis caused by gas-forming organisms.

3. Early diagnosis is to be encouraged in this form of cholecystitis and if this is achieved the treatment of choice is cholecystectomy with antibiotic therapy. If the diagnosis is delayed and the patient's condition is satisfactory, conservative treatment with antibiotics is justified. If the condition settles with conservative treatment, it is important that elective cholecystectomy be carried out, as the persistence of a chronic focus of infection appears to be high.

ACKNOWLEDGEMENTS

I am grateful to Mr. S. F. Reid whose guidance and teaching over the past three and a half years made the successful management of this case possible. The capable assistance of the house surgeon, Dr. Leon Carp, is much appreciated.

I would also like to thank the Assistant Radiologist, Dr. W. Nairn, and the hospital photographer, Mr. J. Scrimgeour, for their considerable assistance in preparation of films.

Permission by the Medical Superintendent, Dr. L. Hudson, to publish this case is gratefully acknowledged.

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METASTATIC OVARIAN CARCINOMA*

ULCERATION AND INVASION INTO THE STOMACH

By J. S. T. Cox

PERFORATION of a benign or malignant ulcer on the posterior gastric wall into the lesser sac is a well recognized occurrence. Penetration into the posterior stomach wall with subsequent perforation from secondary carcinoma in the lesser sac is an unusual and less frequently reported event.

CASE HISTORY

A 34 years old female was admitted following the sudden onset of severe left-sided upper abdominal pain. At the time of admission the pain had been present for six days, was stabbing in nature and relief was obtained only by lying in the left lateral position. There was no shoulder tip pain and no vomiting but some biliary regurgitation had occurred two days prior to admission. Her previous history revealed that in 1956, at the Hospital for Women, Soho, after complaining of pain in the right lower back and groin for six months, she had a right ovarian cyst removed and a left salpingo-oophorectomy. At the same time some nodules were noted on the peritoneal surface of the uterovesical pouch. Subsequent section of the ovary revealed primary papilliferous cystadenocarcinoma.

Examination revealed nothing more than some guarding and tenderness in the left upper quadrant of the abdomen. Rectally nothing was felt, and there was no gross pelvic abnormality on vaginal examination.

Special investigations:—

Serum amylase—88 somogyi units per 100 mls.

White cell count—6,000.

Differentials:

Neutrophils—80 per cent.

Lymphocytes—12 per cent.

Monocytes—8 per cent.

Haemoglobin—79 per cent.

Plain X-ray of the abdomen and chest revealed only some minimal elevation of the left half of the diaphragm. The temperature was 100°F.

Two days later a mass, which was tender, dull to percussion and moved on respiration, appeared in the left upper quadrant of the abdomen. It increased in size over a period of five days reaching to five fingers below the left costal margin and there was transmitted pulsation. Fourteen days later it began to subside and after three weeks was pal-

pable as a firm, slightly tender irregular swelling, extending two fingers below the left costal margin. The temperature, however, persisted between 99° and 100°F.

When the pulse became steady and the mass remained constant in size, a barium meal was performed. The report was as follows:—



FIG. 1. The air in the stomach is brought into relief by the convex margin of the mass projecting into the stomach.

"A straight film was taken which shows the outline of this mass extending into the left upper abdomen with a convex outer margin and extending from D.12 to L.3 (Fig. 1). Following a little barium, the majority entered the stomach, and a large irregular projection was noted, extending from the middle third of the lesser curve wall and this remained constant throughout the whole examination. Food debris was in the stomach, but it was not possible at any time to visualize a normal duodenal cap appearance, yet barium appeared to pass moderately well around the small gut. The lower part of the pyloric antrum fills poorly and shows a little roughening of outline (Figs. II and III). Films taken from two and one-half to twenty-two hours show a considerable residue of barium to remain in

*Received for publication 14th January, 1960.

the stomach. The large projection adjacent to the lesser curve had emptied a little by this time (Fig. IV)."

Since this was inconclusive, a gastroscopy was performed by Dr. P. E. T. Hancock of the Royal Marsden Hospital, and the report on this is as follows:—

"At the junction of the middle and distal third there was a bulge into the anterior wall of the lesser curvature which was persistent, but the mucosa appeared normal. Around this the antrum could be seen but there was no view of the actual pylorus. Folds normal. Mucosa of the proximal portion of the stomach appeared normal. View of the antrum was much limited by the projection into the lumen—noted above—but no definite abnormality detected. No view could be obtained of what appeared to be a perforation on the lesser curvature and the appearance suggested a neoplasm projecting into the lumen of the stomach ? sarcoma."



FIG. II. This shows the irregular projection from the lesser curve, food debris and irregularity of the pyloric antrum, with non-visualization of the duodenal cap.

A gastric mucosal biopsy was done at the same time, however, failed to give any added information.

Tests for occult blood were persistently positive.

The patient's general condition deteriorated slightly whilst in hospital and a laparotomy was performed on 8th May, 1959. This revealed:—

1. A mass of solid fleshy tissue involving the lesser sac from the retroperitoneal region and adherent to the mid-region of the lesser curve

adjoining the posterior stomach wall. Through the anterior stomach wall was a palpable ulcer crater extending into this fleshy mass.

2. The lymph nodes in the prepyloric, subpyloric and suprapyloric regions were involved and enlarged.
3. In the pelvis was a solid palpable mass 4 cms. by 3 cms., mobile and attached to the pelvic mesocolon. This had been impalpable rectally. A biopsy was taken from the subpyloric mass and that in the lesser sac and the abdomen closed.

On month later the patient was alive but some fluid had collected in the peritoneal cavity.



FIG. III. A lateral view of prepyloric and duodenal region in support of Fig. II.

The sections of the initial lesion removed in 1956 have been reviewed by kind permission of the Hospital for Women, Soho, and compared with those of the biopsy specimens removed at operation.

The original sections showed a primary papilliferous cystadenocarcinoma of the ovary with none of the characters of the Krukenberg type of ovarian secondary deposit and the distribution of the growth suggested origin from the ovary itself.

The sections taken from the operation biopsy show several pieces of fibrous tissue and omental fat infiltrated by a strikingly similar carcinoma. The frond-like papilloma and adenomatous spaces

of the primary growth were faithfully repeated and the cellular type was identical. There does not appear to have been any further dedifferentiation.

COMMENTS

The interest of this case lies in the fact that a metastatic carcinoma has ruptured into the stomach, giving a clinical and radiological picture similar to that of a primary lesion of the stomach wall itself.



FIG. IV. At twenty-two hours. There is residual barium in the stomach. Some emptying of the projection and barium in the colon.

In view of the recent history associated with the rise in temperature and neutrophils and the development of a mass which enlarged and then subsided, it was thought this was the result of a small leak from the posterior stomach wall into the lesser sac causing a perigastric abscess.

Then remained the decision as to whether perforation had occurred in a malignant ulcer. It was thought possible that the original primary lesion had been in the stomach, and that in the ovary a secondary, although the pathologist did not agree with this. The other possibility was whether this could be a perforation of a benign ulcer

and unconnected with her previous ovarian pathology. A second radiologist reporting on a second barium meal favoured this. However, Oschner and Graves, 1938, when reporting on a series of 1,500 cases of sub-phrenic abscess, found that only 3 per cent. occurred in the lesser sac. Again this lesion in the lesser sac may have been due, as suggested by Aird (1957), to trauma to the pancreas, acute pancreatitis, tuberculous peritonitis or polyserositis.

A false cyst arising after trauma to the abdomen or acute pancreatitis tends to enlarge between the stomach and transverse colon or above the stomach. However, there was no history of trauma, the serum amylase was never greater than 88 somogyi units, and the incidence of pancreatic cysts is rare as Aldis (1956) in a study of crush injuries to the abdomen, found only 2 cases in a series of 75,000 in-patients treated in the previous fifteen years. The barium meal in the present case showed constant irregularity both of the lesser curve and pyloric region. Where there is a pancreatic cyst the barium meal shows a filling defect in the stomach which can be eliminated by displacement of the stomach or duodenum.

Tuberculous peritonitis and polyserositis were not considered seriously since neither the recent nor past clinical picture were in favour of either diagnosis. Solid tumours of the pancreas had to be considered and these may reach a size when they become palpable. I could find no report of islet cell tumours doing this, but adenocarcinoma of the body and tail may enlarge to this extent, although rarely, and in this position also it may spread to nodes along the border of the gland, to coeliac and to splenic nodes and invasion of the posterior stomach wall is possible.

However, it was a surprise to find on operation that the mass was solid apart from some central necrosis where the barium meal had passed, extra-gastric and involving the stomach secondarily. Lymphosarcoma of the retroperitoneal nodes has been reported to have invaded the duodenal wall, necrosed and produced a similar X-ray picture (Heriot and Edwards, 1959).

SUMMARY

It would appear that secondary spread from the carcinoma of the ovary had occurred along the peri-ovarian lymphatics to involve the para-aortic lymph nodes from which extension had occurred into the lesser sac. This only became apparent when invasion and erosion of the mid-region of the stomach wall occurred, giving a picture similar to that of perforation from a chronic gastric ulcer. The X-ray films display the crater in a translucent area which is, in fact, solid secondary ovarian adenocarcinoma.

ACKNOWLEDGEMENT

My thanks to Mr. R. C. B. Ledlie for allowing me to publish this case and for his many helpful suggestions.

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LYMPHANGIO-ENDOTHELIOMA OF THE ARM FOLLOWING RADICAL MASTECTOMY FOR CARCINOMA OF THE BREAST*

By R. J. RIDDELL

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IN 1948, Stewart and Treves described a previously unrecognized complication of a long-standing oedema of the arm following radical mastectomy.

Since this time many cases have been described, mainly in American literature.

Two cases of this syndrome have occurred at this hospital in the past three years.

CLINICAL SYNDROME

Stewart and Treves, in their original article, described the clinical and pathological features of the syndrome exhaustively and further papers on the subject have not improved on their description.

In all their cases there had been an immediate post-mastectomy oedema of the arm near the operation site. This occurred without any complication in healing, or any post-operative infection, or any sign of thrombosis. The swelling had persisted and had gradually extended from the arm to the forearm and the hand. Pain was absent, but increased swelling caused discomfort due to skin distention. Pain due to peri-arthritis and myalgia occurred with increased swelling. Ultimately, in these oedematous limbs, muscular movement caused severe pain, so that the limb was held in a fixed position and this in itself increased the oedema. Attacks of erysipelas were common; hyperkeratosis and telangiectasis also occurred.

After an interval of years (average seven), purplish red, sub-dermal, slightly raised, macular or polypoid lesions appeared. The primary site was the arm or the antecubital fossa. The lesions occurred as a solitary

tumour, followed by satellite nodules which sometimes became confluent. These lesions later became partially bullous. The larger lesions became papillomatous and covered with a shiny tense epithelium with a tendency to ulceration and discharged a serosanguineous exudate. The lesions tended to heal and break down again. New lesions appeared and all stages between imminent ulceration, discharge and healing were present. Fresh nodules appeared on the forearm, the dorsum of the hand and on the adjacent thorax.

The skin became pachydermatous, with pitting oedema. The nodules became flattened and showed on section a soft greyish-red parenchyma. The larger nodules became necrotic, leaving a deep sloughing ulcer. Haemorrhagic discharge was not a feature. These nodules did not resemble the cutaneous nodules of recurrent breast carcinoma. They did not appear initially in the operative field and fresh groups of tumour nodules appeared centrally rather than peripherally.

The tumour nodules continued until treated. The patient frequently succumbed to metastatic growth. In Stewart and Treves' cases the metastases were pulmonary.

These authors recommended radical forearm amputation. In spite of this radical surgery, further tumour nodules reappeared on the chest wall at the site of the amputation in many cases. Metastatic lung tumour terminated the patient's life in nearly every case. In their experience the tumour was not sensitive to radiotherapy.

CASE REPORTS

Case 1

Mrs. D.L.M., housewife, aged 54 years, had a radical mastectomy for a carcinoma of the left breast in June, 1947. She had a course of post-operative X-ray therapy.

*Received for publication 22nd February, 1960.

About December, 1948, the left arm became increasingly oedematous, which was unrelieved. In 1954 a condition similar to the clinical signs described by Stewart and Treves was noted in the left arm, and a diagnosis of lymphangiosarcoma was made. A course of X-ray therapy was given to the left arm with some improvement. In 1955 tumour nodules recurred and extended over the arm. Amputation of the left arm was performed. The patient remained well for some months but towards the end of the year commenced to feel tired and was unable to do any of her usual tasks. One month before admission to hospital she developed pain in the lower left chest and was found to have a large effusion in the left pleural cavity. X-ray at this time was alleged to show secondary deposits in some of the ribs on the upper left side. She had a further course of X-ray therapy. Chest aspiration on several occasions gave several pints of dark fluid blood. On one occasion aspiration was followed by injection of Triethylenemelamine (T.E.M.) 10 mgms. in 10 ccs. of distilled water. The patient became increasingly dyspnoeic and developed congestive cardiac failure. She died on the 4th June, 1956.

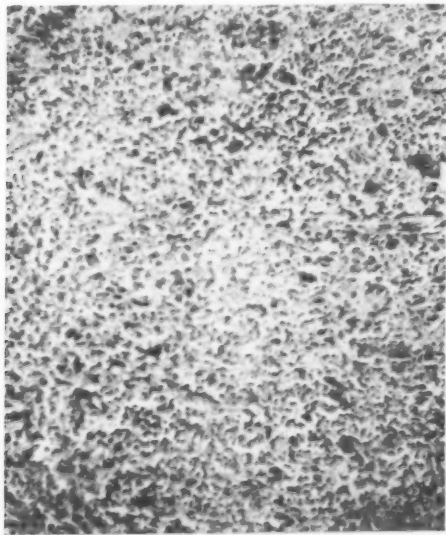


FIG. I. Mrs. D.L.M. Section of the tumour showing the undifferentiated character of the tissue with small irregular tissue spaces lined by tumour cells.

Post-mortem examination showed a well healed scar of the radical mastectomy over the left chest. The atrophic skin here was covered with numerous fine telangiectases but there were no tumour nodules. The amputation scar was well healed and there was no evidence of tumour nodules on the skin surface of the thorax.

In the left chest, the pleural cavity contained many pints of turbid brown heavily blood-stained

fluid. This contained a large amount of blood clot and the left lung itself was compressed against the mediastinum. The parietal pleura was covered with nodular friable tumour growth. This consisted of soft haemorrhagic tissue and was arranged partly in flat sheets where the nodules were confluent and partly in closely packed individual nodules up to 0.5 cm. in diameter. The dome of the diaphragm was covered with a thick mass of homogeneous encephaloid tissue 3 cms. thick. A similar thick sheet of homogeneous tissue covered the surface of the visceral pleural over the lung. There were no tumour deposits in either lung.

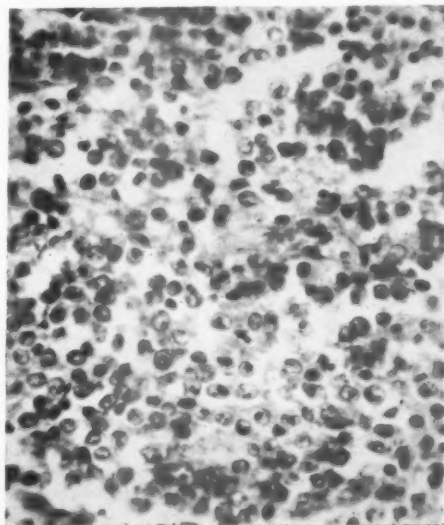


FIG. II. Mrs. D.L.M. High-power view of tumour.

On the left side the parietal pleura was easily stripped from the rib surface, carrying the tumour tissue with it. The ribs appeared normal. However, there were large irregular nodular tumour masses in the third and fourth intercostal spaces in the scapular line. These consisted of elongated raised nodular masses of soft haemorrhagic encephaloid tissue. In the third space this was 3 cm. long; in the fourth space it was 8 cm. long. The masses eroded the rib margins to some extent on either side but no tumour tissue was found in the ribs themselves.

Microscopic examination of the tumour tissue (Figs. I and II) showed a poorly differentiated structure. Polygonal cells were arranged loosely on strands of loose reticular tissue. In places the tumour cells were thought to be lining spaces, but at that time these were regarded as more of an artefact than a tubular tissue space. The tumour was regarded as a sarcoma of the reticulum cell type rather than a recurrence of the breast tumour.

However, in Stewart's and Treve's article the Figure 9 illustrating the anaplastic lymphangio-

sarcoma appears quite similar in structure to this tumour. The tumour has now been classified as a lymphangio-endothelioma.

Case 2

Mrs. L.P., housewife, aged 84 years, had a left radical mastectomy in 1937. She had a history of swelling of the left arm since the mastectomy 23 years before.

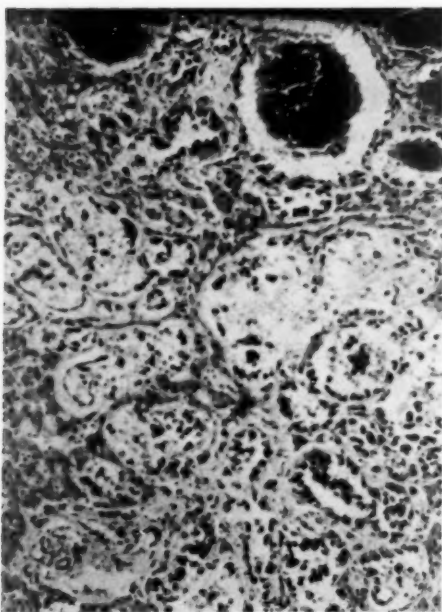


FIG. III. Mrs. L.P. Section of the tumour metastases in the liver to show the well-differentiated character of the tumour tissue.

Two years ago the left arm showed increasing swelling. One year ago she had noticed small lumps which appeared in the skin of the outer, anterior and inner aspects of the forearm. These were at first pink, and as they increased in size they became purplish and "like a blister." The most recent lesions appearing had been larger and painful. She was examined in January, 1956. There was a left radical mastectomy scar which was soft and elastic and without evidence of tumour recurrence. The left arm was oedematous and over the inner, anterior and outer aspect of the middle of the arm was a zone of closely packed purplish skin nodules. These were macular, raised, with a shiny atrophic epidermal covering. Two nodules on the outer part of the arm were larger, about 2 cms. in diameter and raised 1 cm. above the skin surface. Many had a central ulceration.

Biopsy of a skin nodule at the Peter MacCallum Clinic was reported: "Section showed a normal epidermis with a grossly oedematous dermis. There

were many dilated vessels and some haemorrhage and blood pigments. Polymorphs were numerous. Throughout this tissue were groups of malignant epithelial cells having large hyperchromatic nuclei. Mitoses were fairly frequent. Summary: secondary carcinoma—consistent with primary breast carcinoma." On review the conclusion was reached that this was a lymphangiosarcoma.

With X-ray therapy the skin nodules subsided. The patient was kept under observation and skin nodules recurring from time to time were treated with X-ray therapy, which seemed to keep the lesions under control.

In May, 1959, she was admitted to hospital with severe ascites. By paracentesis several pints of deeply blood-stained fluid were removed. Examination of this fluid showed several small clumps of malignant cells with nuclear degenerative changes. She was given radio-active chromium phosphate into the peritoneal cavity.

In June, 1959, the patient was admitted to the Austin Hospital with dyspnoea and severe ascites. She had been coughing blood-stained mucus for a week. Paracentesis was performed and five pints of heavily blood-stained fluid were obtained. Malignant cells were not found on examination. The peritoneal fluid re-accumulated with great rapidity. The patient's course was rapidly downhill and she died on 13th June, 1959.

Post-mortem examination showed a well-healed scar of the radical mastectomy on the left side. There was a mild degree of pitting oedema of the left forearm and hand, but none in the right arm. No tumour nodules were found. The cervical nodes on the left were normal but in the right cervical chain were two large well-encapsulated tumour nodules 2 cms. in diameter, which were not fixed. Section showed densely haemorrhagic tissue with the appearance of blood clot. In the chest, the pleural cavities were normal. In the right lung there was a single large rounded tumour mass 6 cms. in diameter, extending from the hilum into the superior segment. The tumour was of cavernous appearance and deeply haemorrhagic. It appeared to merge gradually at the periphery with the surrounding lung tissue. Proximally, tumour tissue had infiltrated the bronchial wall to form a flat white plaque on the mucosal surface 2 cms. in length. In the posterior mediastinum and around the lung hilum there were metastatic tumour deposits up to 3 cms. in diameter, well encapsulated, fairly well demarcated and consisting of deeply haemorrhagic tissue. The peritoneal cavity was filled with many pints of deeply blood-stained fluid. There was a saccular aneurysm of the anterior lumbar aorta, just above the bifurcation, but the wall here was firm and fibrous. There was a huge tumour mass on the hilum of the left kidney approximately 10 cms. in diameter, well encapsulated and showing a white tissue of cavernous appearance whose spaces were filled with blood clot. The liver was not enlarged and the surface was smooth. Section showed tumour nodules scattered throughout the parenchyma (Fig. III). These were about 4 cms. in diameter and had

a markedly cavernous appearance, the spaces being filled with blood clot. Tumour nodules were clearly demarcated from the liver parenchyma.

Microscopic examination of the tumour in the thoracic and abdominal viscera showed a remarkably well organized neoplastic tumour whose cells were polygonal with large vesicular nuclei arranged loosely on a fibrous reticulum. These tumour cells were sometimes in small groups but frequently were clearly differentiated into a vascular structure. In the more specialized areas, large spaces filled with blood were conspicuous. This was marked in the liver metastases where differentiation was of a high order and the tumour cells formed large vessels, few of the cells being not organized in this fashion (Fig. IV).

The tumour clearly had an entirely different appearance both macroscopically and microscopically from a breast carcinoma.

DISCUSSION

Treves (1957) recently reviewed the complication of lymphoedema after radical mastectomy. He stated that at the Memorial Hospital between 1939-1943, of 1,007 patients with carcinoma of the breast, 768 were classified as having primary operable breast cancer and of this number, 319 had swollen arms. A swollen arm occurred in about 41 per cent. of patients with primary operable breast cancer.

The syndrome described by Stewart and Treves must be rare but these authors considered that many cases are not recognized. Its true incidence was probably higher. The comparatively high incidence of oedema of the arm following operation is a factor tending to increase the occurrence of lymphangio-endothelioma.

Stewart and Trevis considered that a systemic carcinogenic agent acting over a long period on the chronic oedematous arm was the aetiological factor. The syndrome could perhaps be compared to a carcinoma arising in a chronically discharging sinus from an osteomyelitis of a limb bone.

Treatment of the condition is not satisfactory. Stewart and Trevis regarded the tumour as not sensitive to radiotherapy and recommended interscapulo-thoracic amputation as the immediate treatment of choice. However, the tumour lesions recurred on the chest wall and all these patients died with pulmonary metastatic deposits.

More recently Southwick and Slaughter (1955) and Nelson and Morfit (1956) have treated patients with X-ray therapy.

The former authors' case has survived five years with no sign of recurrence. The latter authors combined X-ray therapy with local tumour excision and skin grafting.

Radical amputation has had no success and these less radical procedures seem the treatment in favour at present.



FIG. IV. Mrs. P. Liver sectioned at autopsy showing cavernous appearance of the metastatic tumour.

SUMMARY

Two cases of lymphangio-endothelioma of the oedematous arm following radical mastectomy for breast cancer are reported.

In one case amputation of the arm was performed. In the second case the local lesions were treated with X-ray therapy.

Both cases terminated fatally within two years with metastatic tumour deposits in the viscera.

ACKNOWLEDGEMENT

I wish to thank Dr. Kaye Scott, radio-therapist to the Austin Hospital, for the opportunity of presenting these two cases.

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CHRONIC ULCERATION AND CARCINOMA*

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CHRONIC ulcers of several years' duration following extensive burns, surface injuries or radiation damage were at one time common clinical entities. With the present-day understanding of soft tissue healing and those active measures which are now used to ensure its early and effective accomplishment, it is less common to see ulcers of long standing and the complications which may accompany them.

In the Plastic Surgery Unit of this hospital however, there have been enough cases of malignancy supervening in chronic ulcers (Marjolin's ulcer) to lend weight to the old adage that a person with a persistent ulcer has only to live long enough for it to become malignant. Most of these malignancies have been squamous cell epitheliomata but there have been occasional cases of basal cell carcinoma and of sarcoma arising in chronic ulcer. We are surprised sometimes by a histological diagnosis of malignancy in old ulcers which had been excised when there was no apparent clinical diagnosis of this condition, suggesting that some ulcers had already undergone malignant change before the usual and obvious clinical changes had developed.

On these observations, it was thought fit to pose two questions, "Is the onset of malignancy in a chronic ulcer a slow ingravescent process?" and "Are there any histological criteria which would indicate the incipient onset of malignancy?". With this in view chronic ulcers of one or more year's duration, which for various reasons had been excised, were studied to see if there were any particular histological features relating to the age of an ulcer which may be of premalignant significance.

MATERIAL

The study is based on a review of 50 cases of chronic ulcer treated in the Unit over the period 1946 to 1958. Forty-five of these cases had no clinical change suggesting

malignancy, while the remaining 5 cases had some macroscopic change suggestive of malignancy. Radionecrotic ulcers following X-ray treatment for malignant skin lesions were excluded from this review because of the possibility of residual carcinoma confusing the diagnosis but radionecrotic ulcers following X-ray therapy for benign skin lesions have been included.

The duration of the excised ulcers varied from one year to more than forty-five years, but in many cases the original lesions were of longer duration. These were as follows: burns 17, injury 13, radiotherapy 12, circulatory disturbances 5, syphilis 1, and unknown 2.

The distribution of the ulcers was widespread with 29 on the lower limbs, 11 on upper limbs, and 10 on the head, neck or trunk.

HISTOLOGY

Sections of excised tissue from all these 50 cases were examined. A histological diagnosis of malignancy was made in 12 cases of which 2 could be regarded as borderline. The remainder (38 cases) showed no histological evidence of malignancy, of the 12 malignant ulcers 5 had been diagnosed clinically but in the remaining 7 the diagnosis was made only on microscopic examination.

The epithelium and underlying tissues about all these ulcers were examined in detail for any variation from normal and assessment was made of the following features: (1) the marginal epithelium, (a) keratin on the surface (b) the prickle cell layer (c) the degree of elongation of the rete pegs and any irregularity of their outline (d) the width of the Malpighian layer and its intensity of staining (e) variation in characteristic of cell nuclei; (2) the base of each ulcer (a) the fibrosis present and (b) the amount of inflammation.

A summary of these features is presented in Table 1.

*Received for publication 13th January, 1960.

TABLE 1

		Malignant	Non-malignant
Ulcer margin			
Keratin	increased	9	29
	normal	3	9
Malpighian layer	widened	11	25
	not widened	1	13
Paler staining	present	8	24
	absent	4	14
Nuclear variation	present	2	1
	absent	10	37
Elongation of the rete pegs	present	11	25
	normal	1	13
Irregularity of rete pegs	present	3	6
	normal	9	32
Ulcer base			
Fibrosis	heavy	5	9
	light	7	29
Inflammation	severe	5	19
	mild	7	19

(a) Keratin

Most of the marginal areas in each group had excess of keratin so that this feature was regarded as having no significance as a pre-cancerous change.

(b) Malpighian layer

It was found that in surrounding scars and in the margins of the ulcers the epithelium was occasionally thinned and its lower border was flattened (Fig. I). In most cases however, the epidermis as a whole was thickened and hypertrophic. Near the region of any malignancy the Malpighian layer was generally widened and frequently it stained with less intensity but even these changes were not constant.

(c) Rete pegs

There was always some elongation of rete pegs in the malignant cases but again this change was also present in many of the non-malignant cases. Gross irregularity of the rete pegs in surrounding areas (Fig. II) was present in only 3 of the 12 malignant cases.

(d) Inflammation and fibrosis

The degree of inflammation showed no significant variation in either malignant or non-malignant ulcers. In general it was more obvious in the non-malignant group. The degree of fibrosis was notably denser in relation to the malignant ulcers but this would be expected in the more chronic lesions.



FIG. I. The common appearance of scar tissue about the margin of chronic ulceration. Thin epithelium with absence of the normal rete peg structure covers dense fibrous tissue. There is increased keratin on the surface.

To summarize, ulcers which had become malignant showed in the adjacent epidermis more elongation and irregularity in the rete pegs, widening of the Malpighian layer and generally a greater degree of fibrosis in the

ulcer base. The degree of keratinization and staining showed no significant variation. None of these features in the epithelium, however, would clearly distinguish malignant from non-malignant ulcers.

Further analysis of the non-malignant ulcers in relation to their age did not show any significant difference in the Malpighian layer or in the rete pegs.

DISCUSSION

Ulcers in old burn scars have provided the most frequent source of Marjolin's ulcer in this series. Of 17 cases of chronic ulceration in old burn scars, malignant change was present in 6 cases. In the available literature it is significant that of 190 cases of scar cancer quoted by Treves and Pack (1930) 17 per cent. were in burn scars and that Schrek (1941) has estimated that 18 per cent. of skin cancers he studied had arisen in old burn scars.

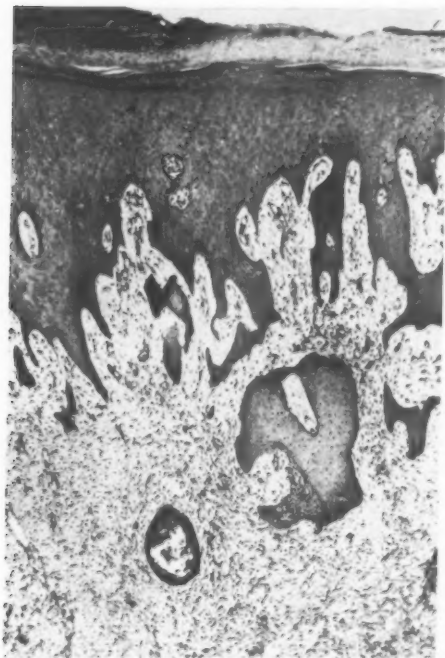
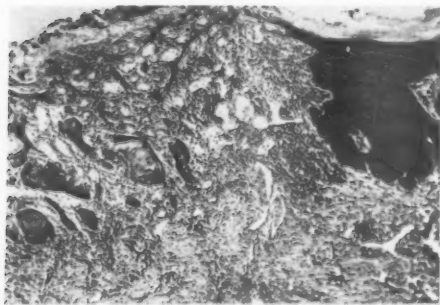


FIG. II. Hypertrophic scar in the region of a chronic ulcer showing gross irregularity of the rete pegs. This appearance, though seen about the margin of many malignant ulcers, is not characteristic.



(a)



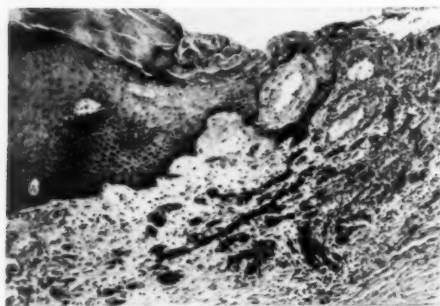
(b)

FIG. III (a) and (b). Sections taken from the marginal region of malignant ulcers in scar tissue. Though hypertrophic the epidermis is fully differentiated and extends right to the edge of the ulcer. There is no transitional state between fully differentiated epidermis and malignant invasion.

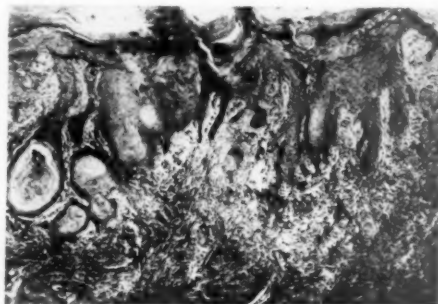
Radiodermatitis has been the other common source of chronic ulceration and subsequent malignancy. The incidence of malignant change in radiodermatitis and radionecrotic ulcers has been estimated by Saunders and Montgomery (1938) as 10 per cent. in their series of 259 cases of radiodermatitis. Leddy and Rigos (1941) reported 26.2 per cent. malignant change in 80 cases of radiodermatitis. Teloh (1950) recorded 28.1 per cent. in 121 cases and Macomber (1951) an incidence of 17.4 per cent. Although the number of cases of radiodermatitis in this study is too small for comparison, it was found that 5

We do not subscribe to this view as we observed this change in the epithelium at the side of the tumour in only one-third of the malignant cases and a similar irregularity in the epithelium in one-quarter of the non-malignant ulcers (Fig. III [a] and [b]).

No features of the epithelium adjacent to the chronic ulcers which we studied gave an indication that tumour formation would be found elsewhere. Nowhere was any change observed that could be regarded as premalignant, if the 2 cases where the alteration in epithelium was regarded as borderline are excluded (Fig. IV [a] and [b]).



(a)



(b)

FIG. IV (a) and (b). The borderline appearance between that of chronic ulcer and malignant ulcer. The irregular downgrowth of epithelium is not orderly as in Fig. II yet the cell characteristics are not obviously malignant and the downgrowth is not rapidly out of position.

out of 12 such ulcers showed malignant changes.

It has been pointed out by Routledge (1959) that there is a considerable risk of development of malignancy following treatment by radiation and that unfortunately the treatment of benign lesions by radiation also incurs this risk. In the present series of 12 radionecrotic ulcers with 5 instances of malignancy following radiation for a benign lesion, the initial indication was a very doubtful one. This included radiation treatment for paronychia, hyperkeratosis and for fibro-adenoma of the breast. Basal cell carcinomata have followed radiation for superfluous hair on the chin and one of the histologically suspect lesions was in a radium burn. We would therefore join in decrying the indiscriminate use of radiation therapy for benign lesions.

Routledge has described irregularity of the rete pegs as a definite premalignant change.

In a comparison of the malignant and non-malignant ulcers, the only significant difference appeared to be a greater degree of fibrosis which could simply indicate a longer period of ulceration.

In short, these findings would agree with those of Lawrence (1952) that no regular or typical transitional stage is found between the hypertrophic epithelium of chronic ulceration and frank neoplastic invasion.

SUMMARY

1. The history and histology of 50 chronic ulcers which had been excised in a Plastic Surgery Unit were studied.
2. Ten showed definite malignant changes and 2 more showed changes suggestive of malignancy.
3. The length of time for which ulceration had been present over one year did not

appear to be a significant factor in histological appearances.

4. No changes in the epithelium about chronic ulcers were found which could be regarded as premalignant.
5. The long term danger of radiation is suggested by the fact that no less than 5 of the tumours in this small series developed in ulcers which followed radiotherapy for benign lesions.

ACKNOWLEDGEMENTS

I wish to thank Mr. B. K. Rank, Head of the Plastic and Faciomaxillary Unit at The Royal Melbourne Hospital, for allowing me access to his Unit records. I also wish to

think Dr. J. D. Hicks for his help and guidance in this project.

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A HAND-OPERATED DIATHERMY FORCEPS FOR GENERAL SURGICAL USE*

By J. G. BROCKIS

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FOR a long time surgeons have met with some inconveniences when using diathermy machines. A foot-switch controls the apparatus and usually operates at mains potential. This has an uncanny way of worming itself under the legs of the operating table and its connection frequently becomes damaged. For these reasons an insulated dissecting forceps has been made which incorporates a hand-control. It can be used to seize bleeding points as well as to touch artery forceps already applied.

both sides of the circuit and any residual high frequency current is bypassed by connecting two 0.25 micro-farad condensers centre tapped to earth across the ends of the coils. Experimentally it was found necessary to insert a small high voltage capacitor in the diathermy lead itself to prevent peaking of the circuit by the high frequency chokes, and to earth one side of the bridge rectifier through a 300 ohm resistor. It is essential that a screened transformer be used from which the 6-8 volts AC is obtained.

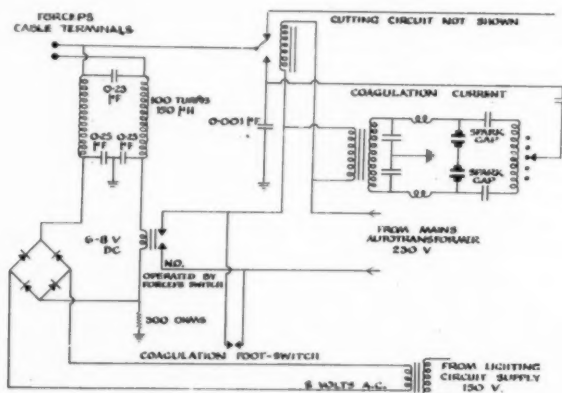


FIG. I. The circuit described in the text for use with the hand-operated diathermy forceps.

Essentially the circuit (Fig. I), which can be simply fitted, consists of a relay mechanism operated by 6 volts D.C. obtained by full wave rectification of the lighting voltage found on most diathermy machines. In order to prevent the relay being operated by the high frequency diathermy current, an inductance of 150 micro-henries is incorporated in

A prototype insulated forceps and a diathermy needle (Fig. II) have been constructed in the workshops of the Royal Perth Hospital and are made of linen-impregnated plastic sheet within which the metal connections are carried. The plastic has been bonded with Araldite which has been found to withstand repeated boiling.

*Received for publication 7th March, 1960.

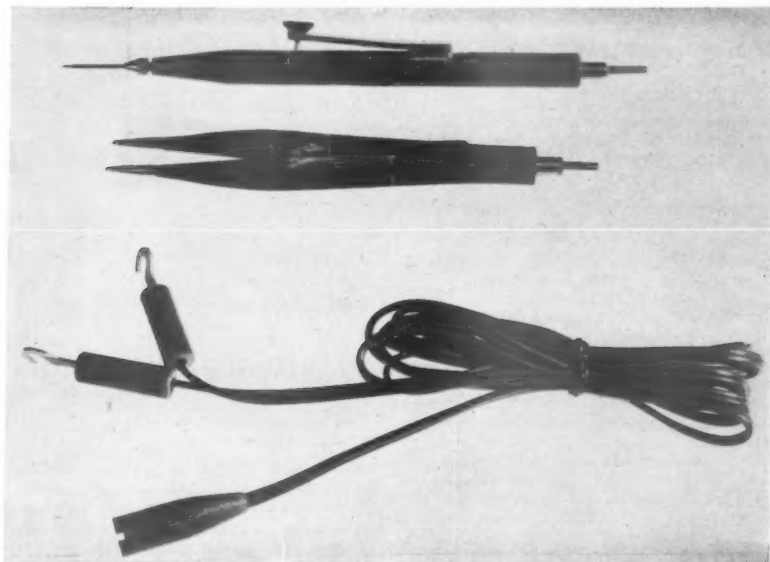


FIG. II. The forceps, diathermy needle and the plastic covered connections.

ACKNOWLEDGEMENTS

The modifications to the standard diathermy apparatus described above were made by Mr. P. Higgins of the Western Australian State X-ray Laboratories Service, and the writer is also indebted to Mr. J. F. Quigley and Mr. L. Young of the Instrument Repair

Department of the Royal Perth Hospital for making the prototype forceps illustrated above.

The diathermy forceps are now available from Messrs. T. H. Spicer and Son, 29 Wiloughby Road, Hornsey, London, N.8.

Books Reviewed

SURGERY IN INFANCY AND CHILDHOOD.

By MATTHEW WHITE, F.R.C.S.(Ed.), and WALTER M. DENNISON, F.R.C.S.(Ed.). Edinburgh and London: E. & S. Livingstone Ltd., 1958. 8½" x 5½", xii plus 444 pp., 266 illustrations. Price: 45s. (stg.).

It is a fact which occasions considerable surprise that although paediatric surgery has become a recognized specialty throughout the world, no textbooks in English have been available for teaching the subject until recently. This is so in spite of the constant flow of literature in surgical journals dealing with the specific problems which relate to surgery in infants and children.

The aim of the authors has been to remedy this omission and to produce a handbook for senior medical students and general practitioners using the wealth of clinical material which they have acquired during the past thirty years in Glasgow where paediatric surgery has long been taught as a specialty and included in the final examination in surgery through a compulsory question in the written paper as well as in a clinical examination.

With such a vast subject it has been difficult for the authors to keep the size of the book within bounds. Although freely and well illustrated with excellent photographs and line drawings they have however succeeded in supplying a statement on most paediatric conditions in a book of less than 450 pages. This has been achieved by an economy of words and leaving out all discussion on surgical techniques, or controversial hypotheses regarding the aetiology of congenital abnormalities.

While the book is thus excellent for the student, it is not of great value for the surgeon requiring a reference book or more detailed information.

In saying that, one is not decrying the book but simply pointing out that the purpose of the authors has been to produce a handbook for students, and this they have achieved. What is said is clearly stated and with its contents most paediatric surgeons would be in agreement.

THE ANTI-GLOBULIN (COOMBS) TEST IN LABORATORY PRACTICE.

By I. DUNSFORD and JEAN GRANT. Edinburgh: Oliver & Boyd Ltd., 1959. 8½" x 5½", 120 pp., xi tables, 9 figures. Price: 12s. 6d. (stg.).

In an era of auto-immune disease, it is important that everyone associated with medicine should have some knowledge of the Coombs test.

Introduced originally as an aid in blood grouping, its principles have now been generalized into aspects of medicine and medical research which range from colitis to cancer.

The introduction emphasizes the amount of literature on the test and for the bibliography alone, the book is worth its price.

Its presentation is lucid and sufficient diagrams are given for full understanding.

There is some reiteration — mostly concerned with cold incomplete antibodies in normal refrigerated blood. This is probably only a matter of careful emphasis and links with the author's insistence on complete controls at every stage of the test.

The later chapters on applications and oddments are a fair indication that this is a technique which will continue to expand.

This book gives a lot of knowledge in regard to an important subject in a small space.

SURGERY OF REPAIR AS APPLIED TO HAND INJURIES.

By B. K. RANK and A. R. WAKEFIELD. Second Edition. Edinburgh and London: E. & S. Livingstone Ltd., 1960. 10" x 7", 284 pp., 219 figures. Price: 45s. (stg.).

Surgery of Repair as Applied to Hand Injuries, by Rank and Wakefield, now appears in its second edition. The first edition was published in 1953. The second edition has no radical changes. In their preface to this new edition the authors write, "a few modifications and amplifications have been indicated by further experience."

The text has a clear, authoritative style, leaving the reader in no doubt as to what is intended. The illustrations, nearly all photographs, are numerous and for the most part are clear. This has obviated the need for line drawings which have proved a popular form of medical illustration.

No statistics are given and although this makes for easier reading, the authors might, in a future edition, give consideration to the addition of an appendix containing some of the more important data. Undoubtedly a tremendous amount of analysis has preceded the publication of this book and it seems a pity that it was not inserted in some unobtrusive way. Statistics represent useful information for a reader critical enough to try and reach his own conclusions rather than accept those already formed for him. Statistics also represent accurate statements of fact and often show that impressions formed are wrong.

The title of the book suggests a highly specialized monograph suitable only for plastic surgeons. But all surgeons are concerned with repair. All students must understand the principles involved. Part two of the book, dealing with the primary treatment of hand injuries, is so well written that it could be made compulsory reading for any medical undergraduate. A surgical post-graduate will find the whole book an excellent aid.

So clearly have Rank and Wakefield described the features of the surgery of repair that the reader cannot but regret that the authors "... have resisted ... the temptation and repeated suggestions that we extend the subject beyond the confines of the title to other ramifications of hand surgery."

The book is a tribute to the publishers, E. & S. Livingstone.

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